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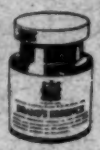

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A FURTHER STUDY OF THE TEETH OF 5-YEAR-OLD CHILDREN IN RESIDENTIAL HOMES AND DAY SCHOOLS

BY

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AND

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with the Assistance of JOAN JOYNER, B.Sc., and MARION KELLEY

It was brought home to one of us in 1929, when examining the teeth of some 5-year-old children in London County Council schools, that there was a great difference between the condition of their teeth and those of children living in orphanages in the Midlands. Among the L.C.C. pupils at this time there were only 4.7%* with caries-free or almost caries-free deciduous dentitions, whereas it was obvious at a glance that the number of orphanage children with caries-free teeth was much higher. In 1943 the chance arose of making a limited number of systematic inspections of the teeth of children living under different conditions. It was decided, in the first place, to see whether any changes had occurred in the teeth of L.C.C. pupils between 1929 and 1943. The improvement was so great (the 4.7% of 1929 had increased to 24.2% in 1943) that similar inspections were made in 1945, 1947, and 1949 (Mellanby and Coumoulos, 1944, 1946; Mellanby and Mellanby, 1948, 1950).

It was not until 1945 that an opportunity arose of seeing whether there was any real connexion between living conditions and dental health in orphanage and L.C.C. children. Concurrently, a similar inquiry was made among pupils attending private (fee-paying) day schools; like the L.C.C. children, they lived with their own families. Meanwhile various workers had observed a tendency for children brought up in orphanages to suffer little from caries, while others, including Collett (1935) and Schiötz (1939) in Norway, and Anderson *et al.* (1934), in Toronto, had published a few figures relating to mixed age groups indicating that there was less caries among such children than among the general population. The threefold survey of 1945 (Coumoulos and Mellanby, 1947) showed that children living in homes had the worst dental structure, suggesting poor calcifying diets during tooth development, and yet had the least caries—less than the children of comparatively well-to-do families and much less than the L.C.C. group. It would have been instructive had we been able to parallel every survey in L.C.C. schools with similar inspections among residential homes and private-school children, but this was not possible. Early in 1950, however, the two groups in question were reinvestigated. This seemed especially urgent, since the present policy of boarding-out and adoption is removing nearly all 5-year-olds from homes.

In this paper we are using the term "home" or "residential home" to cover residential nurseries and schools administered by the State or by voluntary bodies; previously we called them institutions. These are maintained for children deprived of a normal home life for a variety of reasons, such as the death, prolonged illness, or prison sentence of one or both parents, illegitimacy, or lack of suitable housing facilities. Formerly such children were accommodated in orphanages, or institutions of various types, including workhouses.

*This figure of 4.7% included children with up to three teeth with very slight caries (C, cavities) since the number of caries-free individuals was negligible.

Clinical Material and Methods

So far as possible the same homes and schools were included in the 1950 survey as previously, but others from near-by districts were added to increase the size of the sample. In all, 55 homes (mainly situated in London and the surrounding country, with a few in the Midlands) and 32 private and independent schools (in London and adjacent counties) were visited, the total numbers of children dentally inspected being 542 in the former and 560 in the latter group. In 1950, as in 1945, the average age of both groups of children was approximately 5½ years.

The standards adopted for hypoplasia and caries in the clinical examination, and the methods used in calculating the results, were the same as those employed on many previous occasions. The degree of hypoplasia was estimated with a right-angled probe: and the rougher the labio-buccal surfaces the greater the degree of M-hypoplasia. For diagnosing caries an illuminated mirror and sharp rat-tailed dental explorer of standard size were used. The teeth were neither dried nor *x*-rayed, since, although these aids to detection would have resulted in more caries being seen, the findings would not have been comparable with those of past surveys; moreover, the diagnosis of the presence and extent of caries by any known method often comes in for criticism. For this type of work the important thing is to have agreement on the standards to be adopted, followed by adherence to these standards on the part of the investigators, who must work in close collaboration.

The method of presenting the findings, especially those relating to caries, is also open to criticism. We have considered the arguments for and against our system and the schemes adopted by some other workers, but for various reasons we prefer to retain our own. Most dental research workers concentrate mainly on the incidence of disease, including evidence of past decay, in recording the dental status of any given group of the population. They often ignore the structure of the teeth, apart from the grosser forms of defect, and its bearing on the liability to caries. We, however, feel strongly that both should be considered, and that, in accordance with the precepts of preventive medicine, the emphasis should be on the well-calcified and the caries-

free teeth rather than merely on the incidence of defect or disease. In giving the results of the surveys, therefore, we mention first the more ideal state, which it is hoped more children will ultimately attain.

In assessing the quality of the teeth, division was made into those which were externally smooth (no hypoplasia) and those with varying degrees of roughness of the labial or buccal surfaces. M-hypoplasia (King, 1940) was graded M-Hy_1 to M-Hy_3, according to the increasing severity of the defects, while teeth with certain well-defined types of surface irregularity were, as before, classified separately as gross or "textbook" hypoplasia. The numbers of teeth in the various categories of hypoplasia are expressed in the tables as percentages of the total number of teeth examined. Any teeth which were so carious or had such heavy deposits of tartar that the surface structure could not be gauged were omitted from this classification, and thus from the tables dealing with structure. The average extent of surface texture defects of the M-type is represented by the average hypoplasia figure (A.H.F.); this, in conjunction with the percentages of hypoplastic teeth, is, we believe, useful in assessing the improvement or otherwise in the dental structure of successive samples of children—for example, 5-year-old L.C.C. pupils examined between 1943 and 1949.

Caries is expressed both as incidence and as extent for each type of tooth, and for the sum of all types, in each group of children. The incidence of decay by itself, whether given as a percentage or as the D.M.F. (decayed, missing, and filled) figure, can be misleading, since a group of individuals with small cavities would appear in the table to have the same amount of caries as a group in which the cavities were large. For this reason the extent of caries in each decayed tooth is described as being of grade 1, 2, or 3, according to the estimated size of the cavity in relation to the size of the tooth. The average caries figure (A.C.F.), like the A.H.F., is some fraction of 3, and measures the estimated average extent of caries in the teeth. In our opinion the advantage of this method as against those involving tooth surfaces is its relative simplicity; also the results so expressed are comparable with those previously given in this series of reports and those published by others using the same method (Deverall, 1936; Davies, 1939; King, 1940; Coumoulos, 1946; Stones *et al.*, 1949).

In all the surveys made since 1943 it has been possible to calculate from the published figures the D.M.F. values. For instance, the percentage of carious teeth, as expressed in Table IV and in earlier papers, is equivalent to the D.M.F. per 100 deciduous teeth, while the total number of carious teeth divided by the number of children and then multiplied by 100 gives the D.M.F. per 100 children. Since the deciduous incisors are in process of being shed between the ages of 5 and 6 years, the missing ones are considered, in all our surveys, to have been lost naturally, and are therefore omitted from the tables. On the other hand, any absent canines and molars would almost certainly have been extracted for caries at this period; they are therefore included in the caries totals (grade C_3). It will be seen from Table I that the majority of missing incisors in this age group are lower centrals, in which there is very seldom any decay, so that the total number of decayed teeth which might have been present in those counted as "shed" must be small. It is obviously more difficult to analyse caries figures for children of mixed age groups, especially those between 7 and 12 years, because the times of eruption and shedding vary widely. For the 5-year-old children the task is relatively simple. From Table I it is also seen that nearly all the teeth are still *in situ*, with the exception of the lower central incisors, which are normally shed relatively early.

TABLE I.—*Percentages of Teeth Present at Time of Inspection*

Type of Tooth	Residential Homes		Private Schools	
	1945	1950	1945	1950
Upper:				
Centrals ..	94·0	93·3	93·5	94·9
Laterals ..	97·8	98·6	99·2	98·7
Canines ..	99·8	99·9	100·0	100·0
1st molars ..	99·5	99·4	98·2	98·1
2nd molars ..	99·8	99·4	99·5	99·2
Lower:				
Centrals ..	85·7	81·5	76·0	76·3
Laterals ..	98·5	98·9	97·4	97·7
Canines ..	100·0	100·0	99·9	100·0
1st molars ..	98·6	98·9	95·4	95·5
2nd molars ..	99·2	98·6	98·4	98·2
All types ..	97·3	96·8	95·8	95·9

Results

Table II shows the percentages of children in the two groups who were caries-free and those who were nearly so, together with the numbers of D.M.F. teeth per 100 children.

TABLE II.—*Children Caries-free or Almost So, and the D.M.F. Values Per 100 Children in Residential Homes (R.H.) and Private Schools (P.S.)*

Group Investigated	No. of Children Examined		% Caries-free		% Almost Caries-free		% Caries-free + Those Almost Caries-free		D.M.F. per 100 Children	
	1945	1950	1945	1950	1945	1950	1945	1950	1945	1950
R.H.	325	542	57·2	58·7	8·9	14·9	66·1	73·6	204	139
P.S. ..	371	560	50·4	40·0	4·3	18·0	54·7	58·0	275	246

Although the figures for caries-free children are given here to conform with the reports of all the earlier surveys of the series, it has throughout been realized that where the differences between the various groups under review are not spectacular a better picture is gained by considering teeth rather than dentitions. Thus some 10% more private-school *children* had dental decay in 1950, yet the total percentage of caries-free *teeth* (C_0) was actually greater than in 1945. In other words, the disease was more widespread at the time of the later survey, but was of a milder form (see Tables II and IV).

The findings relating to the teeth as opposed to the whole dentitions of the children are given in Tables III to VI. In considering them, a comparison will first be made between the 1950 and the 1945 results for each of the two groups of children separately. Then the results for the residential homes and the private-school groups will be compared. Passing references to the findings in L.C.C. schools in 1949 and 1945 will also be made, although the interval between examination was six months less for them than for the other groups.

Surface Structure of the Teeth

Residential Homes in 1950 and 1945.—It will be seen from Table III (column 4) that 42.0% of all the teeth were perfect or nearly perfect in structure—that is, hypoplasia-free—in 1950 as compared with 32.3% in 1945. This improvement was found in all types except the upper first molars, which remained the same as before, and the upper second molars, where the

percentage decreased. (It is realized that tartar deposits found on a proportion of upper molars, especially second molars, may mask the actual surface texture, thus making it difficult to assess the structure and therefore any change. This applies to all our surveys.) There was very little M-hypoplasia of the most severe grade ($M\text{-}Hy_3$) in any type of tooth in 1950, less than 1% of all the teeth examined falling into this category, as compared with the slightly higher figure of 1.7% in 1945. The A.H.F. had decreased for every type of tooth except apparently the upper second molars, the total having fallen from 0.92 to 0.76. Gross hypoplasia, though relatively low in both series, was lower in 1950 than previously, 1.7% of all teeth, as compared with 4.8% in 1945, being affected by this form of defect.

Private Schools in 1950 and 1945.—Among these children the dental structure was substantially the same in 1950 as in the earlier survey. There were 52.3% of the total teeth free from hypoplasia as compared with 54.0% previously (Table III, column 4). Some, such as the upper incisors and canines, showed improvement in the five-year-period, while others, notably the molars, were not as good as formerly.

As in the case of the residential homes, very few teeth had really severe structural defects ($M\text{-}Hy_3$), the

TABLE III.—*Comparison of Tooth Structure in Residential Homes (R.H.) and Private Schools (P.S.)*

(1) Type of Tooth	(2) Group	(3) Total No. of Teeth Examined for Structure		(4) Good Structure (Hy_0)		(5) Slightly Defective Structure ($M\text{-}Hy_1$)		(6) Defective Structure ($M\text{-}Hy_2$)		(7) Very Defective Structure ($M\text{-}Hy_3$)		(8) Gross Hypoplasia (G-Hy)		(9) A.H.F.*	
		1945	1950	1945	1950	1945	1950	1945	1950	1945	1950	1945	1950	1945	1950
Upper:				%	%	%	%	%	%	%	%	%	%		
Central incisors	R.H.	609	1,001	32·2	51·7	39·2	34·5	20·5	11·6	1·6	0·2	6·2	2·0	0·91	0·59
	P.S.	689	1,053	51·8	63·5	30·9	30·3	14·2	5·4	0·6	0·0	2·5	0·8	0·63	0·41
Lateral incisors	R.H.	635	1,062	32·9	43·3	53·5	46·6	8·3	8·1	0·5	0·2	4·6	1·8	0·75	0·65
	P.S.	722	1,096	64·4	65·0	25·3	32·1	6·5	2·4	0·6	0·0	1·8	0·5	0·42	0·37
Canines ..	R.H.	647	1,079	30·0	49·3	57·2	44·8	9·1	4·4	0·5	0·0	1·4	1·4	0·79	0·54
	P.S.	735	1,113	63·8	75·7	31·2	23·1	3·3	0·7	0·3	0·0	1·0	0·3	0·39	0·25
1st molars ..	R.H.	639	1,070	7·7	7·6	36·9	49·0	46·6	40·3	2·7	0·8	6·1	2·3	1·47	1·35
	P.S.	725	1,095	24·8	12·5	47·2	60·0	23·2	27·2	2·9	0·1	1·9	0·2	1·04	1·15
2nd molars ..	R.H.	644	1,070	6·2	3·0	32·6	36·5	51·7	53·3	5·4	5·1	4·0	2·1	1·59	1·62
	P.S.	736	1,105	19·8	7·2	51·4	49·8	24·9	42·4	2·8	0·5	1·1	0·0	1·11	1·36
Lower:															
Central incisors	R.H.	552	869	78·1	93·3	20·5	6·4	0·4	0·2	0·0	0·0	1·1	0·0	0·21	0·07
	P.S.	559	845	92·5	97·3	7·1	2·7	0·2	0·0	0·0	0·0	0·2	0·0	0·08	0·03
Lateral incisors	R.H.	636	1,064	66·8	76·5	31·3	22·4	0·8	0·7	0·0	0·0	1·1	0·5	0·33	0·24
	P.S.	718	1,088	87·9	86·3	11·1	13·5	0·4	0·0	0·0	0·0	0·4	0·2	0·12	0·14
Canines ..	R.H.	647	1,079	50·1	64·1	40·2	32·3	2·2	1·3	0·0	0·0	2·3	0·8	0·48	0·36
	P.S.	736	1,118	81·8	81·0	13·7	17·9	1·6	0·2	0·0	0·0	0·7	0·4	0·18	0·18
1st molars ..	R.H.	631	1,068	16·6	21·0	40·4	48·1	30·7	27·5	1·9	0·4	10·3	3·0	1·20	1·08
	P.S.	698	1,067	37·0	21·0	41·5	62·6	18·1	16·2	2·1	0·0	1·1	0·2	0·85	0·95
2nd molars ..	R.H.	637	1,064	8·8	20·4	40·0	56·1	36·4	19·5	4·2	0·8	10·5	3·1	1·40	1·01
	P.S.	718	1,094	24·2	22·8	44·6	62·3	26·7	14·2	2·8	0·6	1·7	0·1	1·08	0·93
Totals ..	R.H.	6,277	10.426	32·3	42·0	39·5	38·3	20·9	16·9	1·7	0·8	4·8	1·7	0·92	0·76
	P.S.	7,036	10,674	54·0	52·3	31·0	36·1	12·1	11·1	1·2	0·1	1·3	0·3	0·60	0·59

*A.H.F. (average hypoplasia figure) = $\frac{\text{Total hypoplasia figure}}{\text{Total No. of teeth examined for structure (excluding those with G-hypoplasia or unclassified hypoplasia)}}$

A few of the teeth included in column 3 could not be classified into any of the grades shown in this table. Therefore the percentages do not add up to 100 in all instances.

TABLE IV.—*Caries Incidence and Extent in Residential Homes (R.H.) and Private Schools (P.S.)*

(1) Type of Tooth	(2) Group	(3) Total No. of Teeth		(4) No Caries (C_0)		(5) Slight Caries (C_1)		(6) Moderate Caries (C_2)		(7) Severe Caries (C_3)		(8) Total Carious Teeth (i.e. D.M.F. per 100 Teeth)		(9) A.C.F.*	
		1945	1950	1945	1950	1945	1950	1945	1950	1945	1950	1945	1950	1945	1950
Upper:				%	%	%	%	%	%	%	%	%	%		
Central incisors	R.H.	611	1,011	89·7	90·3	2·3	5·9	6·2	3·3	1·8	0·5	10·3	9·7	0·20	0·14
	P.S.	694	1,063	88·8	90·6	3·0	3·9	6·5	4·9	1·7	0·7	11·2	9·4	0·21	0·16
Lateral incisors	R.H.	636	1,069	95·0	96·4	1·3	2·2	3·5	1·3	0·3	0·1	5·0	3·6	0·09	0·05
	P.S.	736	1,105	94·2	96·7	2·4	1·1	2·7	1·6	0·7	0·5	5·8	3·3	0·10	0·06
Canines ..	R.H.	650	1,084	97·1	99·2	0·3	0·4	2·3	0·3	0·3	0·2	2·9	0·8	0·06	0·01
	P.S.	742	1,120	95·8	97·8	1·6	0·1	1·9	2·0	0·7	0·2	4·2	2·2	0·07	0·05
1st molars ..	R.H.	650	1,084	86·3	93·5	5·7	1·8	5·5	3·3	2·5	1·4	13·7	6·5	0·24	0·13
	P.S.	742	1,120	79·8	84·5	4·7	4·1	9·7	8·8	5·8	2·7	20·2	15·5	0·42	0·30
2nd molars ..	R.H.	650	1,084	74·6	88·1	13·2	6·4	10·2	4·6	2·0	0·9	25·4	11·9	0·40	0·18
	P.S.	742	1,120	72·4	76·3	9·3	11·8	14·2	10·4	4·1	1·5	27·6	23·8	0·50	0·37
Lower:															
Central incisors	R.H.	557	883	98·9	99·7	0·4	0·1	0·7	0·0	0·0	0·2	1·1	0·3	0·02	0·01
	P.S.	564	855	98·9	99·5	0·5	0·0	0·5	0·2	0·0	0·2	1·1	0·5	0·02	0·01
Lateral incisors	R.H.	640	1,072	99·2	99·4	0·5	0·3	0·3	0·0	0·0	0·3	0·8	0·6	0·01	0·01
	P.S.	723	1,094	99·4	99·6	0·1	0·1	0·4	0·2	0·0	0·1	0·6	0·4	0·01	0·01
Canines ..	R.H.	650	1,084	98·9	99·1	0·3	0·6	0·6	0·4	0·2	0·0	1·1	0·9	0·02	0·01
	P.S.	742	1,120	97·3	98·8	0·3	0·3	2·2	0·8	0·2	0·1	2·7	1·2	0·05	0·02
1st molars ..	R.H.	650	1,084	83·5	86·2	4·2	5·2	7·2	6·5	5·1	2·1	16·5	13·8	0·34	0·25
	P.S.	742	1,120	69·9	71·4	4·5	7·5	14·6	14·3	11·1	6·8	30·1	28·6	0·67	0·56
2nd molars ..	R.H.	650	1,084	73·7	77·9	10·2	13·1	9·2	5·9	7·0	3·1	26·3	22·1	0·49	0·34
	P.S.	742	1,120	65·1	61·1	11·5	18·6	14·3	15·4	9·1	5·0	34·9	38·9	0·68	0·64
All types ..	R.H.	6,344	10,539	89·5	92·8	3·9	3·6	4·6	2·6	1·9	0·9	10·5	7·2	0·19	0·12
	P.S.	7,169	10,837	85·8	87·3	3·9	4·9	6·9	6·0	3·5	1·8	14·2	12·7	0·28	0·22

* A.C.F. (average caries figure) = $\frac{\text{Total caries figure}}{\text{Total No. of teeth (including extractions)}}$

proportion being 0.1% of all the teeth examined, as compared with 1.2% in 1945. The only teeth coming within this grade in the more recent survey were the molars. There was very little variation in the A.H.F. for the different types, and the figures for all types together were the same. Gross hypoplasia was again very uncommon in either year, the total amount in 1950 being 0.3% of all teeth examined—1% less than in 1945.

Comparison of Homes and Private Schools.—It will be seen from Table III that the children in the residential homes in 1950 still had worse dental structure than those in the private schools, 58.0% of all teeth as compared with 47.7% showing some degree of hypoplasia (A.H.F. 0.76 as against 0.59). Since, however, in the former group there had been an improvement during the past five years—that is, the percentage of hypoplastic teeth had fallen from 67.7 to 58.0, a 14% reduction—whereas in the latter group there had been none, the disparity was less.

L.C.C. Schools.—Among the L.C.C. group examined in 1949, although the percentage of hypoplastic teeth had increased somewhat since 1945, being 68.0% as compared with 62.0%, the A.H.F. was much the same, 0.88 compared with 0.91.

As regards dental structure, therefore, these observations indicate a general improvement among the residential homes population since 1945, but little change among private schools or the L.C.C. group. The private-school children had on each occasion the best-formed teeth, while the positions of the other two groups were reversed at the time of the second inspection, the residential homes group having better calcified teeth than the L.C.C. group, though not reaching the standard of the private-school children.

Caries of the Teeth

Residential Homes in 1950 and 1945.—Table IV (column 4) shows that, in spite of the fact that a large percentage of the teeth were caries-free in 1945, there was an improvement during the five years between inspections, the respective figures being 89.5% and 92.8% of the total number examined. This improvement was reflected in each type of tooth, but especially in the molars, and in particular in the upper molars. With regard to disease incidence, there were very few carious teeth in these residential-home children in 1950, only 7.2% of the total number being affected, as compared with the previous figure of 10.5%, which was in any case very low. In passing, it may be noted how small was the proportion of carious teeth graded C_3, remembering that this category includes extractions. The extent of caries, as expressed by the A.C.F., fell in 1950 for every individual type of tooth, the figure for all types together being 0.12, as compared with 0.19 in 1945.

Private Schools in 1950 and 1945.—The private-school children showed a slight improvement in the five-year interval. The total percentage of caries-free teeth rose from 85.8 to 87.3, and each type, with the exception of the lower second molars, showed improvement. The percentages of carious teeth at the two inspections were thus 14.2 in 1945 and 12.7 in 1950. The A.C.F. for each type of tooth in 1950 was the same as or lower than before, so that the total A.C.F. showed a slight reduction (0.28 to 0.22).

Comparison of Homes and Private Schools.—The figures given in Table IV indicate that, as in 1945, the teeth of the residential-home children examined in 1950 still had less disease than those of the private-

TABLE V.—*Incidence of Caries in Teeth with Varying Grades of Structure*

(1) Grade of Structure	(2) Group	(3) Incisors				(4) Canines				(5) Molars				(6) All Types			
		Total No. Examined		% Carious		Total No. Examined		% Carious		Total No. Examined		% Carious		Total No. Examined		% Carious	
		1945	1950	1945	1950	1945	1950	1945	1950	1945	1950	1945	1950	1945	1950	1945	1950
Good (Hy_0) ..	R.H.	1,261	2,603	0·5	0·9	518	1,224	0·0	0·3	250	554	0·4	8·1	2,029	4,381	0·3	1·7
	P.S.	1,970	3,142	0·8	1·4	1,071	1,749	0·8	0·6	758	690	3·0	17·0	3,799	5,581	1·3	3·1
Slightly defective ($M\text{-}Hy_1$)	R.H.	891	1,134	2·7	5·7	630	831	2·1	1·4	956	2,026	8·6	13·3	2,477	3,991	4·8	8·7
	P.S.	523	841	8·8	8·7	330	457	6·4	3·5	1,330	2,557	20·6	22·6	2,183	3,855	15·6	17·3
Defective ($M\text{-}Hy_2$)	R.H.	185	211	21·6	14·7	73	61	[13·7]	[0·0]	1,057	1,503	29·8	12·7	1,315	1,775	27·8	12·5
	P.S.	149	83	[30·2]	[9·6]	36	10	[38·9]	[40·0]	669	1,095	56·8	34·7	854	1,188	51·4	33·0
Very defective ($M\text{-}Hy_3$)	R.H.	13	4	[38·5]	[25·0]	3	0	[0·0]	—	91	77	[58·2]	[18·2]	107	81	54·2	[18·5]
	P.S.	8	0	[75·0]	—	2	0	[0·0]	—	77	14	[89·6]	[42·9]	87	14	[86·2]	[42·9]
Gross (G-Hy) ..	R.H.	80	44	[38·8]	[22·7]	24	24	[0·0]	[0·0]	197	112	19·8	12·5	301	180	23·3	13·3
	P.S.	34	16	[38·2]	[25·0]	12	8	[0·0]	[0·0]	42	5	[38·1]	[60·0]	88	29	[33·0]	[24·1]

Note.—For some grades of structure the numbers of teeth were relatively small. The percentages based on these probably have little significance, and are therefore given in brackets.

TABLE VI.—*Extent of Caries in Teeth with Varying Grades of Structure*

(1) Grade of Structure	(2) Group	(3) Incisors				(4) Canines				(5) Molars				(6) All Types			
		Total No. Examined		A.C.F.		Total No. Examined		A.C.F.		Total No. Examined		A.C.F.		Total No. Examined		A.C.F.	
		1945	1950	1945	1950	1945	1950	1945	1950	1945	1950	1945	1950	1945	1950	1945	1950
Good (Hy_0) ..	R.H.	1,261	2,603	0·01	0·01	518	1,224	0·00	0·004	250	554	0·004	0·11	2,029	4,381	0·004	0·02
	P.S.	1,970	3,142	0·01	0·02	1,071	1,749	0·02	0·01	758	690	0·04	0·24	3,799	5,581	0·02	0·04
Slightly defective ($M\text{-}Hy_1$) ..	R.H.	891	1,134	0·04	0·08	630	831	0·04	0·02	956	2,026	0·12	0·21	2,477	3,991	0·07	0·13
	P.S.	523	841	0·15	0·16	330	457	0·11	0·07	1,330	2,557	0·36	0·36	2,183	3,855	0·27	0·28
Defective ($M\text{-}Hy_2$)	R.H.	185	211	0·42	0·23	73	61	[0·25]	[0·00]	1,057	1,503	0·51	0·20	1,315	1,775	0·49	0·19
	P.S.	149	83	0·56	[0·20]	36	10	[0·75]	[0·90]	669	1,095	1·11	0·59	854	1,188	1·00	0·57
Very defective ($M\text{-}Hy_3$) ..	R.H.	13	4	[0·69]	[0·25]	3	0	[0·00]	0·00	91	77	[1·09]	[0·31]	107	81	1·01	[0·31]
	P.S.	8	0	[1·63]	0·00	2	0	[0·00]	0·00	77	14	[2·17]	[0·93]	87	14	[2·07]	[0·93]
Gross (G-Hy) ..	R.H.	80	44	[0·81]	[0·30]	24	24	[0·00]	[0·00]	197	112	0·39	0·18	301	180	0·47	0·18
	P.S.	34	16	[0·76]	[0·50]	12	8	[0·00]	[0·00]	42	5	[0·86]	[1·20]	88	29	[0·70]	[0·48]

Note.—For some grades of structure the numbers of teeth were relatively small. The A.C.F. based on these probably have little significance and are therefore given in brackets.

school children, 7.2% in the former group compared with 12.7% in the latter being carious. Though in both cases there had been an improvement in the five years, this was proportionately greater in the former (a drop from 10.5% to 7.2% of carious teeth—a reduction of 32.0%—compared with a drop from 14.2% to 12.7% in the private-school group—a reduction of 10.5%), so that the gap between the groups had widened slightly.

L.C.C. Schools.—In contrast to the slight improvement in the above-mentioned groups, the 1949 survey of L.C.C. school-children showed no improvement in the caries position as compared with 1945. The proportions of carious teeth in the two years were 26.5% in 1945 and 26.7% in 1949, while the corresponding average caries figures were 0.55 and 0.54.

So far as caries is concerned, then, the children of the residential homes had fewer diseased teeth than those of the private schools and many fewer than those of the L.C.C. schools at both inspections. It must be emphasized again that the caries incidence and extent both in residential homes and in private schools are so low that improvements are inevitably small.

Relation between Structure of Teeth and their Susceptibility to Caries in 1950

It was found in the latest survey, as in 1945 and all other surveys of this series, that within each of the groups of children the better-formed teeth—that is, those with no hypoplasia—were less liable to caries than the M-hypoplastic ones, and that the more severe the defect the greater the liability to the disease (see Tables V and VI). This is true for individual types (incisors, canines, and molars) as well as for all teeth taken together. To demonstrate this point figures are given for three grades of structure in Table VII.

TABLE VII

Structure	% of Teeth Caries-free (C_0)		% of Teeth with Considerable Caries (C_2+C_3)	
	R.H.	P.S.	R.H.	P.S.
Good (Hy_0)	98·6	96·9	0·6	1·3
Slightly defective (M-Hy_1) ..	91·3	82·7	3·9	10·0
Defective (M-Hy_2)	87·5	67·0	5·9	20·8

There were insufficient numbers of M-Hy_2 teeth to give a statistical result for this group.

In all cases it will be seen that for a given grade of structure there was less caries among the children from residential homes than among those in the private schools. Further reference to this point is made in the discussion.

Filling and Extraction of Carious Teeth

Table VIII shows the considerable increase in treatment for both residential homes and private-school children over the past five years, especially as regards fillings. Much more treatment was recorded in both surveys among the private-schools group than among the children in the homes, approximately three times the number of fillings being present. It must be pointed out, however, that slightly over 50% of the carious cavities in the residential homes were very small (C_1 grading), while fewer (38%) of the private-school cavities were graded as C_1.

TABLE VIII.—*Percentages of Carious Teeth Extracted and Filled*

Group	No. Carious Teeth (Including Extractions)		% Extractions		% Filled		% Total Treated	
	1945	1950	1945	1950	1945	1950	1945	1950
R.H. ..	664	754	2·9	5·6	4·7	11·8	7·6	17·4
P.S. ..	1,019	1,378	6·3	7·3	18·5	29·7	24·8	37·0

Note.—The few teeth treated with silver nitrate are omitted from this table.

Staining of the Surface Enamel

The superficial staining of deciduous teeth, which is often confined to the gingival margin and molar fissures, has been commented on by several workers. In past surveys a note was made of this condition and it was found that those children with definite black or brown staining on few or many teeth had less caries than children with none, or children whose teeth showed definite green staining. In this most recent survey there was very little difference between the caries susceptibility of mouths with black and brown stain and those with none (Table IX); but in this connexion it must be remembered that there is very little caries anywhere. Mouths with green stain appear more susceptible, but the numbers of children with this stain are small. Indeterminate varieties of colour were omitted from the calculations. An interesting point was the large proportion of residential-home children—about 33% at each inspection—who showed brown or black stains, as compared with less than 7% of private-school children.

TABLE IX.—*Amount of Caries in Relation to Superficial Staining of Teeth*

Type of Stain	Group	No. of Children		% Carious Teeth		A.C.F.	
		1945	1950	1945	1950	1945	1950
No stain	R.H.	106 (32·6%)	294 (54·2%)	11·5	6·7	0·21	0·11
	P.S.	246 (66·3%)	458 (81·8%)	12·9	12·9	0·25	0·23
Black and brown stain	R.H.	109 (33·5%)	179 (33·0%)	5·7	5·8	0·11	0·09
	P.S.	25 (6·7%)	27 (4·8%)	2·9	10·7	0·05	0·19
Green stain	R.H.	32 (9·8%)	20 (3·7%)	20·2	14·4	0·39	0·22
	P.S.	64 (17·3%)	28 (5·0%)	22·5	18·7	0·46	0·33

Arrest of the Carious Process

There was an apparent decrease in arrested caries among both groups compared with 1945, but this may have been due partly to the personal factor in examination. Arrest is a gradual process, and it is not always easy to agree on a specific standard and maintain it from year to year. The difference between the two groups, on the other hand, is real, the children in the residential homes showing more teeth with arrested caries, 13.5% of all the untreated carious teeth, compared with only 2.9% in the private-school group. So far as they go, therefore, these figures also indicate greater resistance to the carious process in the residential-home children.

Discussion

The object of the 1950 survey among children in residential homes and private schools was twofold: (1) to find out if the differences observed in 1945 between the two groups were still evident; and (2) to see whether there had been any change in dental status in either of the groups over five years.

In both 1945 and 1950 the structure of the deciduous teeth of the residential-home children was not as good as that of the private-schools group, while their caries incidence was lower. Since 1945 there had been improvement in their dental structure (42.0% of the teeth were perfect in structure, compared with 32.3% in 1945—an improvement of nearly one-quarter) and decrease in

caries (from 10.5% to 7.2% carious teeth), in spite of the fact that at the time of the earlier survey the total incidence and extent of the disease were already very low. The private-schools group, on the other hand, which had comparatively good tooth structure in 1945, showed no improvement, while the decrease in caries in 1950 was very small (14.2% of the teeth were carious in 1945 and 12.7% in 1950). Thus the number of carious teeth found in homes children was reduced by nearly one-third, that in private schools by only one-tenth.

Whether the results given in this report are merely periodic fluctuations cannot be stated. No examinations of these two groups were made in 1947, which was the peak year for caries-free teeth in L.C.C. school-children; it is possible, although unlikely, in view of the already small incidence of the disease, that the findings might have been still better then.

What are the reasons for these differences? A comparison between the two surveys does not suggest that the reduction in decay is due to any variation in the sugar content of the diets or to the amount of dental treatment given. The strict rationing of sweets and sugar during the war ensured little difference in this respect throughout the post-eruptive period for the two groups examined in 1945, and those inspected in 1950 would have had, if anything, more sugar and sweets in their diet. Again, there was more treatment in both the groups seen in 1950 than in 1945, but for both years the percentage of carious teeth treated was much greater in private-school children than it was in the homes children.

There may be many contributory factors, but in discussing the differences between the two groups examined in 1945 it was suggested on the basis of experimental and clinical investigations previously made (Mellanby, 1923, 1934) that "the calcifying properties of the respective diets at different periods, both pre-eruptive and post-eruptive, are an important key to the situation." It was pointed out that, as a whole, the relatively well-to-do mothers were more likely (from financial and other considerations) to follow medical advice, then becoming commoner, to take plenty of milk and some source of vitamin D during pregnancy and lactation, than were the poorer mothers of the children who later entered the homes.

It also seemed likely that the mothers in better circumstances would continue to give their children such foods in the months immediately after weaning. During both of these periods the deciduous dentition would be developing and erupting. In the post-eruptive stage, however, there would often be a change in the respective positions of the children from this point of view. Dietary discipline would tend to be relaxed in the case of children living in their own homes, especially where likes and dislikes of such things as eggs, milk, and cod-liver oil were concerned. In the communal life of the residential homes, on the other hand, fads and fancies would be much less in evidence and regular meals of high nutritional and calcifying quality, including an ample allowance of milk and a daily dose of cod-liver oil, were the routine, as was ascertained in the course of the survey.

Thus it seemed probable that the post-eruptive diet of the relatively well-to-do child might not be as good for the teeth as the pre-eruptive diet, whereas the later diet of the residential-homes child would in all probability be much better than that given during the period of tooth development; in this way the resistance to caries of the teeth of the private-schools group would tend to be weakened, and that of the homes group strengthened.

In general these same relative conditions would, in our opinion, be likely to obtain in respect of the children examined in 1950; but whereas the pre-eruptive and post-eruptive diets of the private-school children had probably remained on much the same level in the interim (thus resulting in a rather similar dental condition), there had been certain important modifications, especially in the pre-eruptive period, in the dietary of the homes children. The uncertain quality of diets taken by expectant and nursing mothers and weaned infants in 1938–41 (affecting the structure of the teeth of children seen in 1945) had given place to the rationing of important foods, plus a system of special food priorities for these vulnerable groups, by 1943–6 (when the teeth of the children examined in the later survey were developing and erupting). This priority scheme should have been of especial benefit to the poorer mothers, the section of the population from which the majority of residential homes children are derived. These priorities were available on ration books, and would in many cases be taken as a matter of routine, especially as the milk could be obtained at a reduced price, or even free, and the cod-liver oil and vitamin tablets were issued free to all.

In addition, more was being done than formerly through maternity and child-welfare services to disseminate knowledge of better nutrition. Some of the children entered the homes at the age of a few weeks or months, and in these cases there would be time for the excellent infant and weaning diets given there to exert their effect on calcification, especially of the deciduous molars. In such homes feeding had become even better than in the early war period, owing to stricter planning and supervision, thus increasing, if anything, the favourable post-eruptive influences of such diets.

The work of Schiötz (1939) and Collett (1935), and a small investigation by King (1946), suggested that the earlier in life the children were admitted to the orphanages and the longer the period of residence, the less the incidence of decay. There was evidence of this nature in the data obtained in the 1945 survey, though it was not published; the results obtained in 1950 are now being analysed from this point of view.

The improved dental structure observed in the residential-home children in 1950 can therefore largely be attributed to the better nutritional status in the period of early tooth development as compared with the similar period for the 1945 group. This would of itself tend to result in a reduced caries incidence; but in regard also to the post-eruptive diet these children leading a communal life would appear to benefit greatly. Not only are the diets in residential nurseries and homes well planned, but under the British rationing scheme there is, and has been since 1942 (Ministry of Food, 1947), actually more of the important foods available per child in these homes than can be obtained for children living with their families. Moreover, in the latter case any special allocation for the children may be all too often pooled with the food for the rest of the household. Furthermore, these children have far more say than those in the homes in regard to what they will or will not eat, and in many instances they are able to spoil their appetite by eating food of low nutritional value between meals.

In our opinion, few families in Britain, whatever their economic status, provide for their children diets which

are as good in their calcifying properties as those served in children's residential homes to-day, and in few is the intake of cod-liver oil and even milk likely to be nearly so regular or so long maintained. It seems to us, therefore, that the excellent diet given in these homes is responsible for their low caries incidence. The reduction noted since the previous survey five years ago is no doubt partially the result of improvement in dental structure, but it may have been enhanced by the even better diets provided for these children in recent years.

It is a challenging thought that, on the whole, children brought up in orphanages and other public institutions have a better chance of remaining free from caries (at least up to the age of 5 years) than children of the same age brought up in private families.

Summary

The new results given in this paper are compiled from the individual dental charts of a survey in 1950 of 542 children aged 5 years living in homes (orphanages and institutions) and 560 children of the same age attending independent and private day schools, mainly in London and surrounding counties.

These two groups of children are compared with similar groups examined in 1945 and with each other, as regards both dental structure (according to the standards originally set up by May Mellanby) and caries incidence and extent. Reference is also made to surveys among L.C.C. school-children in 1945 and 1949.

Among the children in the homes, tooth structure was found to have improved since 1945, there now being 42.0% of the teeth free from both gross and M-hypoplasia as compared with 32.3% previously. Concurrently there was also a reduction in caries incidence from 10.5% to 7.2%.

The private-school group showed much the same average structure in 1950 as before, while the caries incidence had declined only slightly, 12.7% of all teeth being carious as compared with 14.2% previously.

The average dental structure of the residential-home group still lagged behind that of the private-school children, but the difference between the two groups was not as great as before.

In spite of worse dental structure, the children in the residential homes had a lower caries incidence and extent.

The relationship between dental structure and caries in both groups in 1945 and 1950 was such that the greater the degree of M-hypoplasia the greater was the liability of the teeth to caries, but for each grade of structure the residential-home children had less decay than the others. This relation between structure of the teeth and liability to decay has been observed in all similar surveys where the same standards have been adopted.

In each group of children there was an increase in the amount of dental treatment, both fillings and extractions, but particularly the former. At each inspection the percentage of filled teeth among the private-school group greatly exceeded that among the residential-home children.

Approximately 33% of the children from residential homes showed black or brown staining on their teeth in both years, as compared with only 6.7% and 4.8% in the two groups of the private-school children.

Suggestions are made to account for the differences found in both structure and caries incidence between residential homes and private schools, and for the change occurring between 1945 and 1950.

We would like to put on record our grateful thanks to the many people who have so generously assisted us, and without whose help this survey would not have been possible. Particularly we acknowledge our indebtedness to the medical officers and governing bodies for permission to examine the 5-year-olds in children's homes, and to the superintendents, matrons, and teachers who helped with the actual inspections. In the case of the private-school children we thank sincerely all the principals and teachers who so willingly gave us time and assistance to further our researches. We also much appreciated the help given by Miss Irene Allen, of the Medical Research Council's Statistical Department.

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POSTERIOR GASTRO-ENTEROSTOMY IN PEPTIC ULCER: LONG-TERM RESULTS

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(From the Peptic Ulcer Clinic, Western Infirmary, Glasgow)

Gastro-enterostomy in the treatment of peptic ulcer has been the subject of many reports, often conflicting and in recent years seldom favourable. Although to-day abandoned by many, there are still some who champion its cause. In this country Sir James Walton (1950), on careful and complete follow-up of a large series of cases, has found a constant incidence of stomal ulcer of only 2%, but most other workers put the figure much higher, even as high as 40%. The results obtained by Heuer (1944) agree with the experience of the majority of recent writers. In his series of 159 cases followed for periods of 3 months to 10 years (85 followed from 5–10 years) 21.4% developed recurrent symptoms.

In comparing the end-results of gastro-enterostomy and gastrectomy, Heuer found on both short-term and long-term follow-up that the outcome was 10% in favour of gastrectomy; but, as he pointed out, this advantage is reduced by the lesser immediate mortality of gastro-enterostomy. To the debit side of the gastrectomy will have to be added a still undetermined but probably considerable percentage of unsatisfactory results in the form of "small-stomach" syndromes. It is also probably fair criticism of the more extensive procedure that when medical men, and especially surgeons, contemplate operation as a personal issue gastro-enterostomy is given more than fleeting consideration.

With such thoughts in mind, it was felt that a further report on the results of gastro-enterostomy, stressing the long-term results, would be of interest.

Clinical Material

The material in this review consists of 330 patients subjected to gastro-enterostomy for peptic ulceration during the 14 years between January 1, 1924, and December 31, 1937. All cases were under the care of one surgical unit in the Western Infirmary, Glasgow.

All came from Glasgow and adjacent districts. The majority were cases of elective operation. In six cases gastro-enterostomy was performed at the time of closure of a perforation. In two cases gastro-enterostomy with ligation of vessels was performed for haematemesis.

Follow-up

An attempt was made to trace all the survivors and recall them for examination. Altogether 165 patients were traced and were interviewed ; 21, now at a distance in the U.K. or abroad, could be contacted only by letter-questionary. With the aid of the almoner, Red Cross workers, and other voluntary organizations, 50 were traced and found to have died. The information available from their records, relatives, family doctors, and death certificates allowed of some assessment, albeit second-hand. The remaining 70, despite all effort, remained untraced. Many of these patients had lived in an area which had suffered from enemy action. The total number of cases traced, including 24 operative deaths, was 260 out of 330—that is, 78.7%. The investigation was carried out during 1948 and 1949.

It is probably inaccurate to claim this study as a follow-up. Recapitulation would be a more apt term, since many of the cases were being seen for the first time 11–22 years after the event. Where patients were known to have reported to other hospitals at home or abroad, information was obtained from these hospitals.

The distribution of the cases by operation year and their appropriate places in this follow-up are shown in Table I. Table II lists the cases according to diagnosis.

TABLE I.—*Distribution of Operation Survivors by Year of Operation*

Year	Traced Alive	Traced Dead	Untraced	Total
1924	2	2	4	8
1925	6	2	1	9
1926	10	2	6	18
1927	9	1	7	17
1928	12	5	7	24
1929	14	1	4	19
1930	17	5	6	28
1931	22	5	6	33
1932	20	2	6	28
1933	17	7	5	29
1934	21	8	5	34
1935	8	6	5	19
1936	14	0	3	17
1937	14	4	5	23
	186	50	70	306

TABLE II.—*Distribution of Operation Survivors by Diagnosis*

Diagnosis	Traced Alive	Traced Dead	Untraced	Total
Pyloric stenosis ..	19	7	5	31
Duodenal ulcer ..	156	30	61	247
Gastric ulcer ..	11	13	4	28
	186	50	70	306

Assessment of Results.—The results have been divided into (*a*) satisfactory—those entirely free from symptoms—and (*b*) unsatisfactory—those who at any time since operation (1) have had pain of ulcer type ; (2) have suffered from haematemesis or melaena ; (3) have been treated in hospital for medical or surgical treatment of peptic ulceration ; (4) have had *x*-ray evidence of stomal ulcer or reactivation of the original ulcer.

Grades of Severity.—The cases grouped as unsatisfactory were not all poor results. Many, though not cured, benefited by the operation. Accordingly, these results were further graded into: (*a*) severe—pain as severe as or more severe than before operation ; those who had complications or who had required further operation because of peptic ulcer. (*b*) Moderate—pain less severe and more easily controlled by food and alkalis. (*c*) Mild—pain slight, infrequent, and with dyspepsia (flatulence, heartburn, etc.) which required diet restrictions and alkali therapy amounting to some limitation of the normal mode of life.

Failure Curve.—The onset of symptoms after operation has been dated as accurately as possible. The percentage of unsatisfactory results for each follow-up year for the entire series of patients traced (excluding the immediate post-operative deaths) was calculated and plotted in a failure curve. Similar curves were plotted separately for severe, moderate, and mild relapses. It should be noted that, for this study, any patient who had symptoms which amounted to an unsatisfactory result was considered as such from the onset of the recurrence through the remaining years of follow-up: that is, the curve was charted on the basis "once a recurrence, always a recurrence." There was no modification of the curve for those who, for example, had only four years' symptoms in 18 years of follow-up. Similarly, those cases modified by further surgical or medical treatment were regarded as failures from the onset of symptoms. This method no doubt represents a more gloomy picture than a year-to-year assessment would, but in view of the years that had elapsed since operation it was thought that the more simple method was a sufficient tax on the memory of the patient and limited the scope of his imagination.

Results

The relapse rates for years 1–20 have been calculated, but only those of the first five years and for the 10th, 15th, and 20th years are listed in Table III. The failure curves for total, severe, moderate, and mild relapses are shown in Fig. 1.

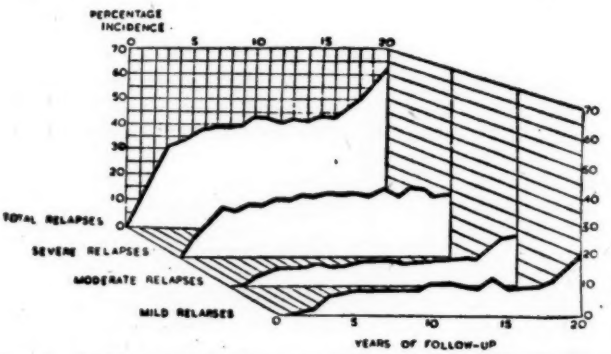

FIG. 1.—Incidence of total, severe, moderate, and mild relapses.

TABLE III.—*Cumulative Incidence of Relapses*

Year of Follow-up	Cases Observed	Percentage Relapses			
		Severe	Moderate	Mild	Total
1	236	8·89	1·27	0·43	10·59
2	228	14·47	4·38	3·95	22·80
3	226	18·14	5·75	7·08	30·97
4	224	17·85	5·81	8·48	32·14
5	221	19·45	6·79	9·05	35·29
10	206	23·30	8·73	11 16	43·19
15	131	25·18	9·22	9·22	43·62
20	40	25·00	17·50	20·00	62·50

It will be seen that the incidence of relapse is most marked in the first five years, 35% having relapsed at the end of that period. Thereafter there is a slower but

steady increase in incidence. In approximate figures it may be said that after a period of between 15 and 20 years 50% have remained symptom-free. Compared with their pre-operative state, 12.5% were markedly improved; 12.5%, though improved, still suffered considerable disability, and 25% were no better or worse. In other words, in 37.5% the results left much to be desired.

Factors Influencing Results

The total relapse rates and the cumulative incidence of severe, moderate, and mild relapses were calculated separately (1) for duodenal ulcer, gastric ulcer, and pyloric stenosis; (2) for males and females; (3) according to age at time of operation; and (4) according to length of pre-operative history.

1. *Duodenal Ulcer, Gastric Ulcer, and Pyloric Stenosis.*—Of the cases traced, 186 were duodenal ulcer, 24 were gastric ulcer, and 26 were pyloric stenosis. It is necessary to define the last-named group, for there are many degrees of pyloric stenosis and it may be organic or spasmodic or both. In this paper cases were assigned to this group when there was *x*-ray evidence of considerable retention, and when copious vomiting, characteristic of gastric delay, was present. Pain was not a marked feature of these cases. The total relapse rates are compared in Fig. 2. In the duodenal group the relapses mount rapidly to 35% in five years. In the subsequent 15 years a further 27% recur. In the gastric ulcer group the large failure rate in the first five years is partly explained by six cases of carcinoma of stomach, the operation having been performed in the belief that these ulcers were simple. In the group with obstruction, though considerably better, the relapse rate is surprisingly high—approximately 25%. However, it was noteworthy that in cases of pyloric stenosis the relapses on the whole were less severe, in marked contrast to the duodenal and gastric ulcer groups, in both of which the incidence of severe relapses was considerable (25% at the tenth follow-up year).

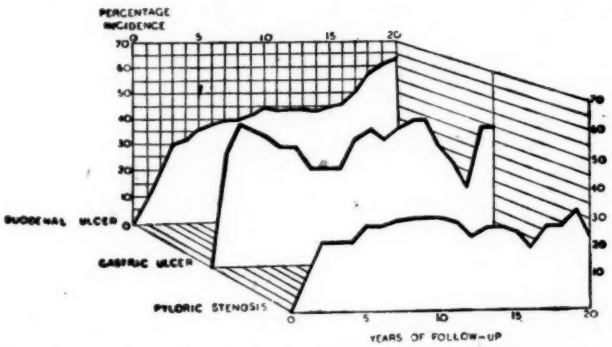

FIG. 2.—Comparison of total relapses in duodenal ulcer, gastric ulcer, and pyloric stenosis.

2. *Sex.*—In the series traced there were 200 males and only 36 females. In Fig. 3 the incidence of total and severe relapses is plotted. In view of the disparity of numbers fair comparison cannot be drawn; however, the impression is gained that whereas the total relapse rates are similar the incidence of severe relapses in females is markedly less.

3. *Age at Operation.*—The cases traced were placed in the following age groups: under 30 (44), 30–39 (81), 40–49 (60), and 50 and over (51). The total relapses are compared in Fig 4. The findings show that the results of gastro-enterostomy are better in the older age groups. Though not recorded in Fig. 4, it was noted that the incidence of mild and moderate relapses was much the same in all age groups. The better results in older patients represent a lower incidence of severe relapses.

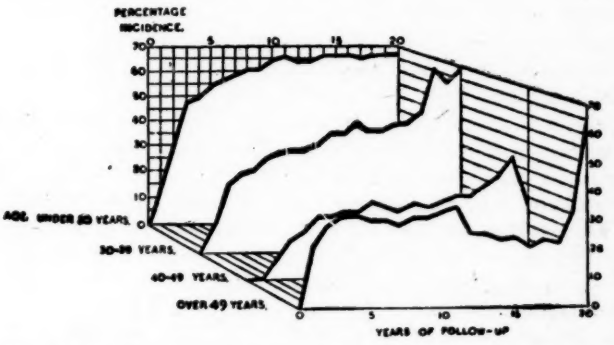

FIG. 4.—Total relapse rates according to age group at time of operation.

4. *Duration of Pre-operative Ulcer History.*—In order to study the relation of the end-results to the pre-operative history, the cases have been allotted to four groups, according to the duration of symptoms prior to operation. The number of cases in these groups is as follows: 1–4 years, 66; 5–9 years, 76; 10–14 years, 57; and 15 years and over, 37. The result is shown in Fig. 5, from which it will be seen that the total relapse rates are closely similar in the different groups. Although not shown in Fig. 5, it was noted that the incidence of severe relapses was considerably higher in the 1–4 years group than in the group of 15 years and over. As an example, at the tenth year the short-duration group gave 30% severe relapses compared with 11% in the long-duration group. The better results in the last group

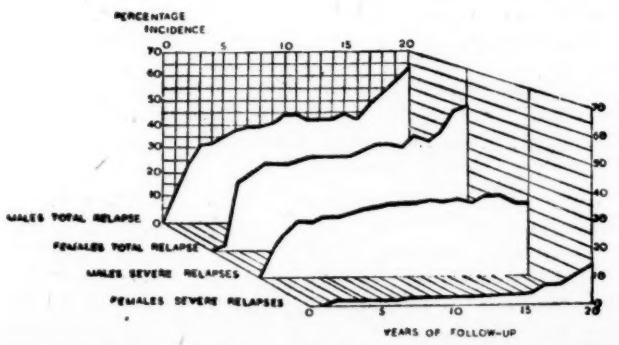

FIG. 3.—Comparison of total and severe relapses in males and females

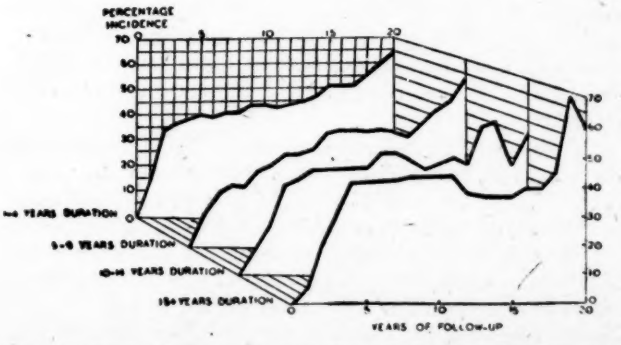

FIG. 5.—Total relapse rates according to years of duration of pre-operative history.

are probably due to the fact that it contains a higher proportion of older patients, of women patients, and of patients with pyloric stenosis. The results in the 5–9 year and 10–14 year groups are similar and intermediate —for example, the severe relapse rates at the tenth year being 27% and 23% respectively.

Incidence of Major Complications, and Late Deaths

Over the total follow-up period, gastro-intestinal haemorrhage occurred in 20 patients (12.2%). There were five perforations in four patients, an incidence of 2.1%. Nine patients (3.8%) found it necessary to undergo further operation for relief of symptoms. Four of the patients already noted as having sustained a haemorrhage also elected to undergo a further operation. Thus the "hard core" of recurrences amounted to 18.1%. Almost two-thirds of these severe relapses occurred within the first five years. No case of gastro-jejuno-colic fistula was encountered in the series traced.

Fifty of the patients traced were found to have died. In the majority the cause of death was unrelated to peptic ulcer. Four patients died from causes directly attributable to peptic ulcer—two as a result of gastrectomy for stomal ulcer, and two as a result of haemorrhage.

In six of the gastric ulcer cases traced the cause of death was carcinoma of the stomach. In only two of these was necropsy performed; in one, malignant change had supervened on a simple gastric ulcer. In the other case necropsy details were not available. Three died within three years, the remainder within five years. Gastro-enterostomy, by leaving the ulcer, carries an immediate risk of mistaken diagnosis and a later risk of malignant change. Carcinoma of the stomach was the cause of death in three cases of duodenal ulcer, death occurring at 10, 12, and 17 years after the operation.

Discussion

The results of this investigation confirm the higher relapse rates reported in the literature. It must be stressed, however, that the method of assessment used has presented the results of the operation in the worst possible light. A year-to-year assessment would no doubt present a more pleasing prospect. Many, though not "cured," were considerably improved. Among those were several who had suffered a complication as an incident in an otherwise uneventful post-operative history. These cases, according to the criteria used, were noted as severe relapses. Similarly, a few cases had a total of only about four years' stomach trouble in as many as 15 follow-up years. Viewed from the vantage-point of "cure" of peptic ulcer by operation, the present results assume a proper perspective. In round figures, after a period of 15–20 years 50% remained symptom-free, and 12.5% whose symptoms were slight were markedly improved. In the remaining 37.5% the operation had failed.

In view of this high failure rate, it seems pertinent to try to obtain a true assessment of the value of the operation. To do so it is necessary to compare the results obtained with those expected without treatment and with those obtained by other methods of operation—for example, gastrectomy. Furthermore, since gastro-enterostomy possesses the cardinal advantage of low initial mortality, the question arises how cases likely to gain permanent benefit by this procedure might be selected pre-operatively.

Comparison with Untreated Cases

Obviously it is not now possible to obtain for study as a control an untreated group of cases strictly comparable with those reported above. Information regarding a somewhat similar group of cases is, however, available from the follow-up studies of Illingworth *et al.* (1946). These studies refer to the progress after perforated peptic ulcer and relate to patients from the same region and of the same social class. The age and sex distribution in the two series are remarkably similar, and by a coincidence the proportion of gastric to duodenal ulcers is almost identical. The two are similar also in that after discharge from hospital their dietary control and anti-acid treatment were generally haphazard. Patients in the gastro-enterostomy group with pyloric stenosis have been excluded, since obstruction is not a factor in the perforation series and would, by inclusion, improve the results of the present series. They differ, of course, in that in the Illingworth series the sole criterion for inclusion was the fact of perforation and in only a small proportion had the symptoms before perforation been severe enough to warrant surgical treatment. These two differences would seem to imply a better prognosis as regards relief of symptoms, and so in comparing the results the scales would be weighted against the present series.

In Table IV the incidence of relapses in the present series and that after perforation are compared at the end of the fifth follow-up year. Fig. 6 shows the year-by-year comparison for the first five years. In Table V and Fig. 7 the incidence of major complications is similarly compared. Here, however, it should be noted that Illingworth *et al.* (1946) arrived at their estimate on the basis of 166 patients followed for the full five years, but used additional information from patients followed for shorter periods.

TABLE IV.—*Five-year Relapses: Comparison with Perforation Series*

	No. of Cases	Percentage Relapses
Present series	197	36·1
Perforation series (Illingworth *et al.*)	166	70·0

TABLE V.—*Five-year Incidence of Major Complications: Comparison with Perforation Series*

	No. of Cases	Percentage Complications
Present series	197	10·5
Perforation series (Illingworth *et al.*)	166+ (See text)	20·0

Compared with the untreated group, gastro-enterostomy has halved the relapses and the complications.

Comparison with Cases Treated by Gastrectomy

Though the results reported for gastrectomy vary, it seems clear that recurrent ulceration is infrequent, not more than 4% (*Lancet*, 1950), and in more extensive ablation of the stomach (Visick, 1948) this complication has been reduced to *nil*. Comparison on this score alone leaves no doubt about which operation is preferable. The position becomes less clear, however, when assessing the importance of those disturbances of alimentary function which are the sequelae of gastrectomy. There is no unanimity in the reported results as regards the incidence, severity, and duration of these complica-

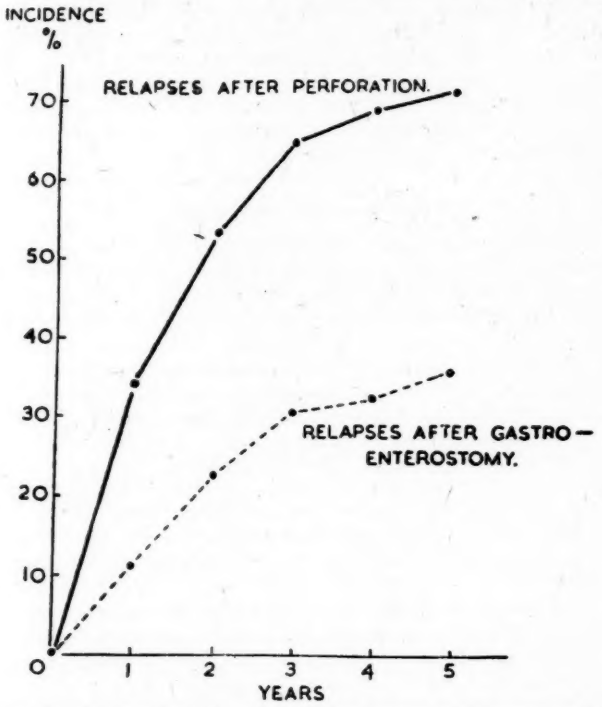

FIG. 6.—Comparison of relapses after gastro-enterostomy with those after perforation.

tions. Muir (1949) found that 75% of patients in his series suffered at some time from the dumping syndrome and that in 34% the patients were considerably handicapped. According to the same author, although this complication sometimes disappeared within three months, not infrequently it persisted for years. That they are better off with their dumping syndromes than with their peptic ulcers is readily admitted by most patients—but not by all (M.D., 1949).

Comparison on the basis of recurrent ulcer is in favour of gastrectomy by a wide margin. How much this margin will be reduced by small-stomach syndromes is as yet unknown.

Selection of Cases for Gastro-enterostomy

The occurrence of good results in at least half the cases has encouraged previous authors to inquire into methods of selection. For example, many surgeons have advocated the use of gastro-enterostomy, particularly in elderly patients with a long history, in women, and in patients of either sex with severe pyloric stenosis. Reference has been made above to the results to be expected in these different categories of cases.

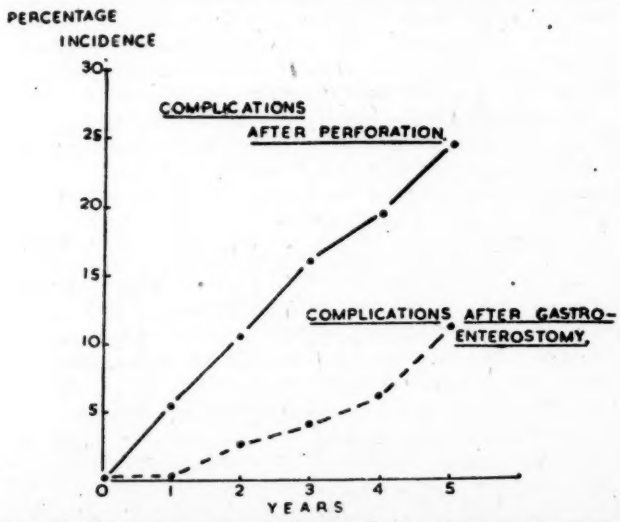

FIG. 7.—Comparison of complications after gastro-enterostomy with those after perforation.

The pre-operative gastric acidity has been used as a criterion by some surgeons on the hypothesis that stomal ulcer is less likely to occur if the acid level is low. Several workers have found this criterion unreliable. For example, Holman and Sandusky (1938) and Nicol (1942) found little relation between the reduction in the acid concentration and the clinical improvement produced by the operation. High pre-operative and post-operative levels of free HCl were consistent with good results. On the other hand, severe relapses were encountered in patients with low acid levels before and after operation.

Taylor (1944) has based selection on gastroscopic appearances and has succeeded in reducing the incidence of recurrence to a low figure. Where the mucosa is very vascular and the rugae exaggerated, relapses tend to occur after gastro-enterostomy. Gastrectomy is advised in these cases.

Walton (1950) is convinced that certain people are more prone to develop gastro-jejunal ulceration. Such patients tend to develop duodenal-ulcer symptoms early in life; there is a high free acid response to a test meal, and both radiography and gastroscopy reveal hypertrophy of the mucosal folds.

Visick (1948) carefully examined stomachs removed because of recurrent ulcer. Abnormally extensive mucosal folds were not a noteworthy feature.

Conclusion

In gastric ulcer, in view of the high relapse rate and the possibility of mistaken diagnosis and malignant change, the operation of choice is gastrectomy.

In duodenal ulcer, the present investigation suggests that the short-circuit procedure has a place in the elderly, in women, and in organic pyloric stenosis. These factors, however, carry no guarantee of permanent cure. With regard to the large proportion of duodenal ulcers requiring operative treatment, until some means of selection is forthcoming, gastric resection will remain the most certain method of cure, an opinion which may require some modification when the true incidence, severity, and duration of post-gastrectomy syndromes are better known.

I am indebted to the late Mr. Roy F. Young and to Mr. George Dalziel for permission to use their records. I also wish to thank Professor C. F. W. Illingworth for his helpful criticism.

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A free booklet, *District Nursing in the Metropolitan Police Area*, gives the names and addresses of all the nursing associations in London. It is available on request from the Central Council for District Nursing, 25, Cockspur Street, London, S.W.1.

IRON ENCEPHALOPATHY

BY

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AND

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The problem of refractory microcytic hypochromic anaemia has been satisfactorily solved in many cases by the intravenous administration of an iron-sucrose complex (Nissim, 1947). Nevertheless, iron by intravenous injection and even by mouth is not entirely without risk. Iron-poisoning was quite common for some years after Pierre Blaud introduced his famous pill in 1832. No further cases were reported until recent years, when, after a period of neglect, the practice of giving inorganic iron salts in large doses by mouth became established again as useful treatment for hypochromic anaemia. Recently, ferrous sulphate by mouth has caused acute gastritis and death from hepatic failure.

Cerebral symptoms following oral iron medication in man are rare. Hurst (1931) reported the case of a very anaemic woman of 58 (Hb 18%) who took 40 gr. (2.6 g.) of iron and ammonium citrate a day. On the 22nd day of treatment (her Hb having risen to 51%) she complained of severe headache and had nine convulsions within 24 hours. Between the attacks she was semi-conscious and had twitching of the right side of the face. She made a good recovery. Beyond saying that the cerebral symptoms resembled those of lead encephalopathy, Hurst gave no opinion on their mechanism, and his title "iron encephalopathy" seems hardly justified on the data published.

Napier (1936) reported a case of presumed iron encephalopathy in an Indian man aged 30 who, after receiving 468 gr. (30 g.) of ferrous sulphate in 26 days for a simple hypochlorhydric anaemia (Hb 18%), had a series of seven fits during a day. He also refers to the case of a woman who had fits following a daily dose of 12 gr. (0.8 g.) of ferrous ammonium citrate for 33 days. Whether the iron had been responsible or not seems doubtful, as the Wassermann reaction was strongly positive.

Mooney (1950) mentions intense headache following an intravenous injection of 100 mg. of saccharated iron.

In the case recorded below severe cerebral symptoms occurred after the intravenous administration of iron.

Case Report

A housewife aged 54, of average mental capacity, attended hospital on January 12, 1949, with a history of increasing dyspnoea and lassitude for three years and of oedema of the ankles and soreness of the tongue for two months. She had no dysphagia. Her menses had been heavy until September, 1947, when they ceased. There was no other history of blood loss. Her diet had been inadequate, though she had not lost weight. There had been no previous illnesses, and she had had two normal pregnancies.

On admission the patient appeared pale. There was no glossitis. The pulse was regular, the blood pressure 160/70. The heart was not enlarged. A soft systolic bruit was audible over the praecordium, but was not thought to indicate valvular disease. No abnormality was found in the lungs, abdomen, pelvis, or central nervous system.

Investigations.—Hb, 41% (6 g.%); R.B.C., 3,400 000 per c.mm.; colour index, 0.6; reticulocytes less than 1%; W.B.C., 7,300 per c.mm.—differential count normal. A fractional test meal showed histamine-fast achlorhydria. A radiograph of the chest was normal. The urine was normal. A diagnosis of hypochromic microcytic (iron-deficiency) anaemia was made.

Progress and Treatment

Intravenous iron therapy was begun, using a solution of saccharated iron oxide (containing 100 mg. of iron in 10 ml.). On the assumption that each 100 mg. of iron should raise the haemoglobin by 4% the total requirement for this patient was 1,500 mg.

On January 14 and 15, respectively, 20 mg. and 50 mg. were given, the injections being made slowly into a vein in the antecubital fossa. On the 17th, after receiving 40 mg. of the iron, the patient complained of dizziness and of tingling in the hands. She also stated that everything appeared "stripy." She then vomited. The injection was discontinued. After this, injections were continued daily, none of them being greater than 75 mg. Only minor complaints of nausea and general malaise were made.

By January 23, the ninth day after the injections were started, a total dose of 380 mg. of iron had been administered. On this day the patient complained of headache and appeared to be very anxious. She later vomited twice. No injection was given. At 7.30 p.m. the nursing staff noted that she seemed strange in her manner and somewhat drowsy. At 9.30 p.m. she had a convulsion, after which she remained deeply unconscious. Her limbs were flaccid and her eyes deviated to the right. Her pulse rate was 120 a minute and the rhythm irregular. There was no pulse deficit. Further convulsions then occurred at intervals of about 20 minutes. Each of these began in the left hand and spread to include the left side of the face, the left leg, and finally became generalized. The C.S.F. pressure was 230 mm., and routine laboratory examinations showed no abnormality. The Wassermann reaction was negative in both the C.S.F. and the blood. Large doses of paraldehyde (16 ml. in all) were given intramuscularly, but the convulsions became more frequent until at 1 a.m. on January 24 they were recurring at five-minute intervals. Thereafter they became less frequent and were confined to the left side of the body. They finally ceased at 4 a.m.

The patient remained unconscious during the next 12 hours, but responded to pinching by groaning or by moving one or other leg. Many minute petechiae were visible over the chest and arms, and bruising had occurred at the sites of pinching. A mild left hemiplegia, affecting the arm more than the leg, was present: this gradually improved during the next three days. Her bleeding-time was 40 seconds; and the clotting-time five minutes; platelets numbered 135,000 per c.mm.; the capillary resistance test (Rumpel-Leede) was positive; and the urine was normal.

By January 28 the patient was conscious and rational. She was able to write and to read a few words correctly, but unable to understand what she read. At 5.45 p.m. she suddenly lapsed into a dreamy state. She sat up in bed and appeared to be completely detached from her surroundings, making no response to the spoken word or to painful stimuli. The few words she uttered (which often included "green" and "yellow") indicated a gross dysphasia. Although this speech defect was associated with a left hemiparesis, she was not left-handed. The only other sign of note was the presence of spontaneous nystagmus. This dreamy state lasted for 36 hours.

By March 8 she was able to read accurately, but a tendency, previously noticed, to read only the right half of printed lines persisted. Her visual fields were apparently full, but co-operation was poor. Her general condition was good, and she was able to walk with assistance. She complained intermittently of occipital headache. The

haematological response to the iron was excellent. There had been a good reticulocytosis, and the haemoglobin rose steadily to 82% (12.1 g.%) on February 2.

She was discharged from hospital on April 1 and returned as an out-patient on April 30. Her haemoglobin was then 92% (13.6 g.%). There were no abnormal physical findings, but she still complained of headache. She was very anxious, and she easily "panicked." Though able to carry out simple household duties, she could not cook a full meal or do any shopping, and was quite unable to make any calculation or to count change. She has remained more or less in the same condition ever since, but has become increasingly depressed at the persistence of her disability, of which she seems fully aware. Her haemoglobin level on June 1, 1950, was 106%.

When last seen on June 24, 1950, she was a little unsteady on her feet. She was depressed at times and tended to be muddled over money. These symptoms were less marked than they were originally. An electroencephalogram at this time showed evidence of bilateral damage in the temporo-parietal regions.

Discussion

Two well-known proprietary preparations have been used many times in other cases without serious mishap. On this occasion both were out of stock, and ampoules of a privately made preparation were offered instead by the pharmacist and accepted for use. It was not fully realized by all concerned that this preparation was other than had been safely used before. It was a solution of saccharated iron oxide prepared from ferri oxid. sacch. equivalent to 100 mg. of elemental iron (Fe) and "apyrogen" (pyrogen-free distilled water) to 10 ml. The saccharated iron oxide was dissolved in the apyrogen with the aid of heat, and the solution was filtered through filter paper while still hot. It was filled into ampoules and autoclaved at a pressure of 10 lb. per sq. in. (0.7 kg. per cm.^{2}) for half an hour. Subsequent analysis of the preparation showed that it contained the correct amount of elemental iron (Fe) and 21.6% of sucrose, the *p*H being 8.7.

Compatibility with serum *in vitro* was demonstrated by mixing 2 ml. of the solution with 2 ml. of normal horse serum, and 0.1 ml. of the solution with 5 ml. of normal horse serum. These mixtures were incubated at 37° C. for one hour and gave no macroscopic or microscopical evidence of coagulation or precipitation. Similar tests with the patient's serum and that of normal human controls were negative. It must not be assumed, however, that precipitation would not occur *in vivo*, and Nissim and Robson (1949) found evidence in mice that a gradual precipitation of proteins did occur *in vivo* and suggested that the compound was broken down gradually as the sugar part of it was metabolized. The LD50 of the sample administered intravenously to mice was approximately 118 mg. per kg., whereas the most suitable preparations were found by Nissim and Robson (1949) to have an LD50 of 300 mg. per kg.

The available evidence does not satisfactorily explain the production of cerebral symptoms. The importance of using pure chemicals in making saccharated iron oxide is emphasized by Robson and Keele (1950), who point out that some of the more transient reactions can be avoided by using analytical grade reagents—for example, "analar" sucrose. Somers (1947) showed that very large doses of iron preparations given by mouth would kill animals, resulting in convulsions and coma for which no adequate organic basis could be found. Cappell (1930) studied the fate of intravenous injections of colloidal solutions of saccharated iron oxide in mice and rats. They did not cause flocculation in contact with plasma, and so pulmonary embolism did not occur. The iron was taken up by the cells of the reticulo-endothelial system, and later formed a loosely bound compound with plasma proteins, through which it was distributed to other organs, notably the liver. Some iron was deposited in the walls of the brain capillaries, but it was rapidly removed, and thereafter no iron was found in the nervous tissues. It is conceivable, however, that capillary damage, sufficient to lead to subsequent injury to the brain, might be caused by some iron preparations.

Polson (1928), in attempting to produce haemochromatosis in rabbits by injecting dialysed iron intravenously, found that pulmonary embolism, presumably from flocculation, was easily caused by long or rapid injections. Ledofsky (1924) injected a 33% solution of saccharated iron several times into eight moribund patients, but no iron could be demonstrated in the brain tissue histologically. Goetsch *et al.* (1946) attributed the toxic effects of iron to a "heavy metal effect," but this term is usually understood to mean the formation of a metallic proteinate when a soluble metallic salt is mixed with a solution of protein. We know that in the present case the preparation used did not produce a proteinate with the patient's plasma.

The analogy of "lead encephalopathy" is not helpful, since the pathology of this state is unknown apart from the findings of arteriosclerosis and meningeal fibrosis in chronic cases.

Summary and Conclusions

A case is reported of severe cerebral symptoms in a woman aged 54 after the intravenous administration of 380 mg. of iron in nine days. The symptoms are tentatively ascribed to iron encephalopathy, but the mechanism of their production remains in doubt. The use of certain proprietary preparations of iron for intravenous use is known to be valuable and fairly free from toxic effects if these preparations have an LD50 for mice of 300 mg. or more per kg. and if dosage is kept below 100 mg. of iron a day. The danger of using unproved preparations is emphasized.

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On May 31, 1950, the Royal College of Obstetricians and Gynaecologists comprised 837 Members and 322 Fellows. Seventy-three of the Fellows belonged to the London division, and Liverpool and Manchester together contained 20, the greatest concentration outside London. Out of 31 Scottish Fellows, Edinburgh contained 11 and Glasgow 14; in Ireland, 9 were in Belfast and 13 in Dublin; while Melbourne and Sydney (Australia) and Montreal (Canada) had 10 Fellows each.

METHOIN IN THE TREATMENT OF EPILEPSY

BY

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During the past five years several new anti-epileptic drugs have become available for clinical use: of these troxidone (" tridione ") is well known in the treatment of petit mal. This paper is a report on a trial of methoin (" mesontoin "; 3-methyl-5,5-phenylethyl-hydantoin), a preparation mainly effective against other varieties of epilepsy. The drug is chemically allied to phenytoin, and is also related to the toxic drug phenylethylhydantoin (" nirvanol ") and to troxidone (Ruskin, 1948)—a fact of some significance when considering its toxic effects. It is dispensed as a pink tablet of 0.1 g.

Clinical trials were first made in 1937 by Boller (1943), who regarded it as valuable but potentially toxic. Results were first published in America in 1945, since when numerous reports upon its use have been made, though no detailed study has appeared in the literature of this country.

Loscalzo (1945, 1946, 1947, 1948), using sodium diphenylhydantoinate (" dilantal ") in a four-year study, reported a 60% reduction of fits in patients suffering from major and minor epilepsy. Toxic effects were few, and the absence of gum hypertrophy and ataxia (common toxic symptoms with phenytoin) was notable. Kozol (1946, 1947, 1950), using doses of up to 1.8 g. a day, treated 200 patients for periods of up to four years, and found a 90% reduction in frequency of fits even though the majority of cases were resistant to phenytoin. Most of his cases suffered from major epilepsy, but the drug was also of use in psychomotor seizures, though not in petit mal. Methoin and phenytoin were sometimes given together, with apparently a synergistic effect; phenobarbitone was not used, as methoin alone exerted a well-marked soporific effect. Harris and Otto (1947) treated 20 cases of psychomotor epilepsy and found that 13 were rendered attack-free. Fetterman and Weil (1947), Fetterman *et al.* (1947), and Fetterman and Victoroff (1949), in a trial with 150 patients, considered methoin to be highly effective in major epilepsy, valuable in Jacksonian epilepsy, and of occasional use in psychomotor attacks. They stressed that good results were achieved when other remedies had failed. In petit mal the drug was not of use. Aird (1948) reported a group of 75 patients resistant to other measures, and found almost complete control in more than half the cases of major, psychomotor, and Jacksonian epilepsy.

Although valuable, it soon became apparent that methoin was also sometimes toxic, and that its use called for the same careful supervision as with troxidone. Gibbs (1947) found that toxic reactions developed in 10% of patients, while of Lennox's (1946) cases 14% had a rash. Kozol (1947) reported a 6% incidence of morbilliform rashes during the first month of treatment, but thought that reactions were made less likely by the use of small initial doses. Loscalzo (1948) stated that a history of previous sensitization reactions was common in patients developing a rash, and stressed the need for a careful history in this respect.

Drowsiness was commonly reported as a side-effect of methoin, but gum hypertrophy and ataxia, which so often occur during phenytoin treatment, were rarely seen.

Minor blood changes were found in 18% of Fetterman and Victoroff's (1949) cases, and Lennox (1946) reported a relative lymphocytosis in 40% of his series. Other authors often commented on the occurrence of a relative lymphocytosis and also of eosinophilia, the latter particularly in the presence of dermatitis.

Serious reported toxic effects included aplastic anaemia and exfoliative dermatitis. In the American literature there are reports of six fatal cases of aplastic anaemia and of eight cases that recovered. The reactions occurred three to eighteen months after starting methoin therapy. Abbott and Schwab (1950), in reviewing the problem, thought that serious blood disorders were not of sudden onset as was held by some, but represented the sudden culmination of a progressive process. They thought it possible that patients developing a sensitization dermatitis were less likely to suffer a serious toxic effect subsequently.

Exfoliative dermatitis is reported in four cases, two being fatal (Ruskin, 1948; Kozol, 1950).

Hepatic disease was found in two fatal cases: one treated with methoin had a central lobular necrosis, and one developed a fatal hepato-renal syndrome following treatment with methoin, troxidone, and phenurone (Levy *et al.*, 1950). Ruskin (1948) states that methoin and troxidone are both potentially toxic, and that both are related to the toxic substance phenylethylhydantoin; combined use of the drugs is therefore dangerous.

Present Investigation

Cases were selected for methoin treatment only after other remedies had been proved ineffective in full tolerated dosage. Patients suffering from petit mal were not given methoin, as published results were so uniformly discouraging. Altogether 41 patients were treated, but only 24 (13 female, 11 male) were considered suitable for assessment; the others either failed to take the drug regularly or omitted to keep adequate records of the numbers of their attacks. The average age was 30, the youngest being 10 years old.

A detailed history was taken in order to determine the type of epilepsy, and careful reference was made to previous drug sensitivity or other form of allergy. Treatment was usually begun with 0.1 g. twice daily, this being increased by 0.1 g. a day at weekly or fortnightly intervals until the necessary amount was being taken.

The number of attacks and the type were recorded at each interview, the frequency being later compared with that before methoin therapy, and improvement being calculated on a percentage basis thus:

$$100 - \left[\frac{\text{average number of attacks a month after treatment}}{\text{average number a month before treatment}} \times 100\right]$$

Some difficulty was experienced in obtaining exact figures of both incidence and type of fit, as most of the patients suffering from major epilepsy had " warnings," or minor psychomotor phenomena, in addition to attacks of grand mal, and tended to neglect the smaller attacks in making their report. Similarly, patients with psychomotor epilepsy were not always conscious of their attacks, or could not be persuaded to keep a list of them other than in their heads. The results are therefore only approximate.

TABLE I.—*Results of Treatment*

Case No.	Sex and Age	Type of Epilepsy	Duration of Methoin Treatment (Months)	Maintenance Dosage (G./Day)	Result (Percentage Improvement)
1	F 36	Major	4	0·9	70
2	F 40	,,	7	2·1	No change
3	F 46	,,	10	0·9	70
4	F 26	,,	6	0·8	100
5	F 29	,,	9	0·9	90
6	M 35	,,	2	0·9	No change
7	M 24	,,	4	1·2	50
8	M 30	,,	4	0·9	50
9	M 33	,,	3	0·3	No change
10	M 25	,,	5	1·2	60
11	F 24	Major and psychomotor	7	1·2	70 + 50
12	F 48	,,	8	0·9	50 + no change
13	M 24	,,	4	0·9	50 + 50
14	M 24	,,	2	0·6	No change + no change
15	M 19	Major and focal sensory	5	0·9	100 + 50
16	F 40	Psychomotor	9	0·6	80
17	F 27	,,	9	1·5	Slight
18	F 41	,,	7	0·9	No change
19	F 10	,,	3	0·1	,,
20	F 23	,,	8	0·6	50
21	F 22	,,	9	0·2	100
22	M 31	,,	7	0·9	95
23	M 21	,,	10	1·2	60
24	M 26	,,	10	0·9	80

Results

The results are shown in Table I. The degree of response to treatment has been divided into three (Table II): much improved, indicating a 70% or more reduction in the frequency of attacks; improved, a 50–70% reduction; and no change if less than a 50% reduction.

TABLE II.—*Degree of Response to Treatment*

	Much Improved	Improved	No Change
Major epilepsy ..	40%	33%	27%
Psychomotor epilepsy ..	31%	31%	38%

It will be seen that best results were obtained with cases of grand mal, but, even so, they are less impressive than most of those reported in the literature. It is possible that selection is to some extent the cause of this: the cases were chosen from amongst a large number of epileptics, the bulk of whom had been attending hospital for some years. Thus there had been every opportunity for a trial of control by other means, and only those in obvious need of something better were given methoin.

A further point is that a certain number of patients, although their attacks were not fully controlled by methoin, stated that the fits were shorter in duration and milder in degree, or that a larger proportion of them did not progress beyond the aura stage.

Illustrative Cases of Successful Treatment with Methoin

Case 4.—A woman aged 26 developed major and minor epilepsy at the age of 10. There was no significant past or family history, and no abnormal signs were observed on examination. She was an unstable person, much upset by her attacks, and had twice attempted suicide. With phenytoin, 0.5 g. a day, and phenobarbitone, attacks were not controlled, and she became ataxic. With methoin, 0.8 g. a day, she was free of attacks and considerably more equable in her behaviour.

Case 15.—A man aged 19, of epileptic stock, had his first attack at the age of 6 months, and by the time he was 11 years old frequent fits were occurring, usually preceded by paraesthesiae spreading up from the right leg to the right arm. There was a left parietal electroencephalographic focus, but air studies were normal. Before methoin treatment he was experiencing one to three major convulsions a week, with also frequent abortive sensory attacks. Phenobarbitone, 4 gr. (0.26 g.), with phenytoin, 6 gr. (0.4 g.), daily, was ineffective and caused ataxia and severe acne. With methoin, 0.9 g. a day, and dextro-amphetamine, 20 mg. a day, no further major attacks occurred, and focal sensory attacks were reduced by half. His behaviour, which for some years had been aggressive and sometimes violent, was not improved.

Case 21.—A woman aged 22 first developed epilepsy at the age of 10, the attacks then consisting of sudden nausea, a bad smell at the back of her nose, and clouded consciousness. In later years she had had up to eight attacks a month of an epigastric aura followed by pallor, semi-purposive hand movements, and sometimes complete amnesia. At times attacks were followed by an indescribably bitter taste, but there were no convulsions. No abnormal neurological signs were found, but there was a right anterior temporal electroencephalographic focus. Phenobarbitone, phenytoin, and troxidone were all ineffective, but only one attack occurred after methoin was given, and complete control has been maintained with 0.2 g. a day. Her eosinophil count rose to 20% during the fourth month of treatment, but she was at that time suffering from a recurrence of hay-fever.

Case 24.—A man aged 26 was knocked out in a boxing match and had his first uncinate attack four weeks later. He was at that time drinking rather heavily. Since then he has continued to have attacks associated with a sweet taste, a sensation of *déjà vu*, and lip-smacking. He can continue working or speaking during an attack, but usually checks his work afterwards to ensure that no mistake has been made. Phenytoin could not be increased beyond 0.6 g. a day, but with methoin, 0.9 g. a day, there is almost full control.

In Cases 21 and 24 methoin exerted its effect rapidly, and it has been noticeable also in other cases of the series that control was achieved early in those responding to treatment, while in those failing to respond large doses were little more effective than small.

Cases Not Responding to Methoin

Case 2.—A woman aged 40 developed epilepsy at the age of 19, the day after her first child was born. There was no family history of epilepsy or any neurological abnormality on examination. The attacks have not been adequately controlled by phenytoin or by methoin, 2.1 g. a day, and the two drugs given together are not more effective.

Case 12.—A woman developed major epilepsy when aged 21. Now, at the age of 48, she has up to 12 attacks a week and there is severe deterioration of personality. Complete investigation, including air studies, has shown no gross abnormality. Some of her present attacks are undoubtedly hysterical, though nearly all are associated with urinary incontinence. With methoin the grand mal attacks are possibly less frequent, but improvement is minimal.

Case 17.—This patient, a woman aged 27, weighed 13 lb. (5.9 kg.) at birth and was cyanosed during the first week of life. She developed normally, however, until the age of 8, when a generalized convulsion occurred. Since then she has continued to suffer occasional grand mal attacks, but now has mainly psychomotor episodes, usually several each day. In them an epigastric aura is followed by a desire to run away, and then masticatory movements occur, she may rub her hands together, and is sometimes incontinent. She cannot speak during an attack. *X*-ray examination revealed a small calcified area in the left temporal lobe, and a slow-wave electroencephalographic focus was present in the same site. Phenobarbitone, phenytoin, or methoin up to 1.5 g. a day has not materially influenced the attack rate.

Electroencephalographic Changes

Little (1949) studied the effect of methoin on the electroencephalogram (E.E.G.) in 26 patients. In 62% an increase in fast activity occurred, and in 58% alpha

activity was decreased. With continued dosage these effects became more pronounced, though there was no correlation between the E.E.G. changes and the therapeutic response. Fetterman and Victoroff (1949) found that in some of their cases the alpha activity became very regular, but they likewise observed no correlation between the E.E.G. pattern and the clinical state. Bercel and Ziskind (1950) stated that methoin led to an increase in fast activity in the E.E.G., and stressed its likeness to the barbiturates in this respect.

In the present series the E.E.G. changes were studied in 18 cases. In 15 there was an increase in fast (20 c/s) activity, sometimes to a marked extent; this did not seem to depend on the duration of treatment, though it disappeared rapidly in one case after withdrawal of methoin.

In eight cases a noticeable effect was the development of a uniform amplitude of all the frequencies. In some the E.E.G. became strikingly regular in appearance, particularly so when 6 c/s activity was prominent in the record. The finding is presumably related to the regular alpha activity reported by Fetterman, though in the present series this frequency was not affected more than others.

A decrease in slow-wave activity was seen in three cases, all of which also developed the increased regularity of pattern.

There was no correlation between the E.E.G. changes and the clinical state. This confirms previous reports, but is none the less surprising in view of the degree of change sometimes produced by methoin. The effect of barbiturates is to some extent comparable in that fast activity may occur during light sedation, but may also persist during coma from barbiturate poisoning. Possibly methoin and the barbiturates have this effect in common because of their related chemical structure, as Bercel and Ziskind suggest, but it seems that the changes are not related to their therapeutic effect.

Methoin Toxicity

In order to avert toxic reactions so far as possible methoin was started in small doses (0.1 g. twice daily) and gradually increased, whilst previously administered drugs were progressively withheld. Phenobarbitone, however, was usually stopped quickly owing to the soporific effect of methoin.

A strict system of control was used, regular blood counts and visits being enforced by giving only a limited supply of tablets. Patients were told to beware of sore throats, fever, skin rashes, malaise, and spontaneous bruising. They were instructed to report any such ill effects at once.

Mild Toxic Effects.—In 83% of the cases the patients complained of drowsiness, which in some continued throughout the period of treatment. In the majority small doses of dextro-amphetamine controlled the symptom fully. Other complaints were dizziness, indigestion, headache, constipation, frigidity, and gain in weight. The last-named occurred in two female patients to a quite marked degree. Gum hypertrophy and ataxia—commonly occurring with phenytoin—were not seen. One patient had had acne before treatment with methoin, and this became worse. A relative lymphocytosis, with or without eosinophilia, occurred in eight patients, and it may be of some significance that four of these gave a history of some form of allergic reaction in the past.

Acute Reactions.—An acute febrile reaction with morbilliform rash occurred in five patients (21%)—two occurred during the first week of treatment, two in the first month, and one in the fourth month. One of these had had an attack of urticaria in childhood, one had had infantile eczema and also developed a rash with phenytoin (but not with troxidone), and one developed a rash with troxidone; two did not give a history of previous sensitivity. Two patients not included in the series also developed rashes: one had had nettlerash once as a child, and the other had suffered from Raynaud's disease of the fingers.

It does seem, therefore, that patients with a history of previous sensitivity should be observed closely for a similar response to methoin, particularly during the early weeks of treatment In all these patients methoin was temporarily stopped, but was gradually resumed without ill effect except in the case reported below.

Severe Reactions

There were three severe reactions, one terminating fatally

Case 19.—This patient suffered from frequent and severe psychomotor attacks and had failed to respond to troxidone or to phenytoin. With the latter drug a severe dermatitis, progressing almost to exfoliation, occurred, and was still evident when methoin was started in small doses. The rash resolved satisfactorily, but methoin could not be continued owing to a progressive degree of leucopenia.

Another patient (not included in the series) was taking 1.2 g. of methoin a day in his fourth month of treatment when he complained of malaise and fatigue. The cervical lymph nodes were enlarged, and there was a petechial eruption over the shoulders. The red blood cell and platelet counts were normal, but there was a relative lymphocytosis of 58%. After methoin had been stopped the rash subsided and the blood picture became normal.

Case 16

A woman aged 40 first had epilepsy at puberty with attacks resembling faints. After several years diurnal and nocturnal grand mal developed, but became infrequent during the last five years, when her main complaint was of distressing psychomotor attacks occurring in series. She had no significant past history, but there was a history of epilepsy in the family. Neurological examination revealed no abnormality.

On October 26, 1949, methoin, 0.2 g. daily, was started, but the patient stopped taking the tablets four days later because of a dazed feeling, this being followed in two days by malaise and a morbilliform rash. On November 9 the drug was resumed and was gradually increased to 0.6 g. a day. This dosage controlled her attacks well and without ill effect except for drowsiness, which responded to dextro-amphetamine.

On January 11, 1950, the patient was well but complained of a gain in weight. On March 11 she complained of soreness inside the mouth, but there was no apparent ulceration and the blood count was normal.

When seen on June 26 she was quite well, but on the night of June 30 she complained of ulceration of the mouth and difficulty in swallowing. The next day a blood count at another hospital revealed 700 white cells per c.mm., all being lymphocytes. She was admitted to that hospital, and examination showed haemorrhagic ulceration of the gums, bruising on the limbs, and a positive tourniquet test. Fresh blood transfusions, penicillin, and "aureomycin" were given, and the condition of her mouth improved. Later, however, severe menorrhagia and a high temperature developed, these continuing until her death on July 9. Serial blood examinations revealed a persisting complete agranulocytosis with scanty or absent blood platelets.

At necropsy the bone marrow was found to be aplastic. There were generalized visceral petechiae and also a recent basal subarachnoid haemorrhage.

It is not known exactly when the blood picture in this case became abnormal, as the illness began when the monthly blood count was due. Previous examination had not shown any abnormality, in particular no lymphocytosis or eosinophilia. When the patient was admitted, however, the granulocytopenia was already severe. She therefore developed a fatal aplastic anaemia eight months after starting methoin therapy. The symptoms were rapid in onset, though it is possible that significant blood changes would have been recognized had more frequent examinations been carried out. There are, however, practical limits to this procedure.

Abbott and Schwab (1950) suggested that serious toxic effects were perhaps less likely to occur in patients developing a hypersensitive reaction early in the course of treatment, but such was not so in this case. It has also been maintained (though not specifically with methoin) that regular blood examinations are fruitless: that by the time changes are evident in the peripheral blood it is probably too late to attempt treatment. An alternative supposition is that blood dyscrasias are of sudden onset and therefore not subject to discovery by regular counts. Abbott and Schwab discounted this, however, and the safest course must remain that of examining the blood as regularly as possible during treatment.

Discussion

As has happened with other new preparations in recent years, methoin proves to be a valuable but sometimes toxic drug. This study confirms its usefulness in major and also in psychomotor epilepsy, and shows that dramatic results may sometimes be obtained. It is, however, a drug of definite toxicity, and its use may be fatal.

Clearly, methoin should be used only when every attempt to obtain control by other means has failed. As beneficial effects usually appear quickly, it is better not to persist with it in patients who are resistant; on the other hand, a short therapeutic trial may be made with little danger.

If methoin is used certain precautions must be routine: (1) Instruction of the patient and relatives on its possible dangers, with the warning that they must report any suspicious symptoms at once. The patient's own doctor ought also to be notified that the drug is in use. (2) It should not be used in conjunction with other drugs likely to cause similar reactions. It should not be used with troxidone, though a number of resistant cases seem to demand this very combination. (3) The patient must be seen and blood examinations carried out regularly throughout the period of treatment.

Summary

The results of methoin treatment in 24 cases of major and psychomotor epilepsy are described. A reduction of 70% or more in fits occurred in 40% of major epilepsy cases, and in 31% of patients with psychomotor epilepsy.

Details of E.E.G. changes due to the drug are given.

Toxic effects, including a fatal aplastic anaemia in one case, are described.

The value and limitations of methoin are discussed.

It is a pleasure to record the help of Dr. Denis Hill, at whose suggestion the trial was undertaken. I am also indebted to Dr. T. V. Mendelsohn for his detailed report on the fatal case, and to the Department of Clinical Pathology at the Maudsley Hospital for carrying out the large number of routine blood tests.

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TUBERCULOSIS IN KENYA

BY

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Details of the Survey

A tuberculosis survey of Kenya Colony was made between February, 1948, and February, 1949. In default of mass miniature radiography, the method of the survey was limited to tuberculin-testing, physical examination, and the examination of sputa. It was possible to get only a single specimen of sputum from each suspect, the likeliest-looking part of which was smeared on a slide, dried, and subsequently examined at the Government Laboratory in Nairobi by the Ziehl-Neelsen method. The usual methods of physical examination were employed, but care was taken to exclude all cases except those with definite and persistent signs. Unequivocal signs of cavitation were taken as a proof of tuberculosis; bronchial breath sounds and post-tussic crepitations were, unless there was evidence of recent acute infection, classified as suspicious of the disease.

Although limited in method, the survey was of considerable extent. Africans were examined at 34 different places, chosen as giving a fair sample of the more populous parts of the colony. In all, 49,812 intradermal injections of 0.1 ml. of 1 in 1,000 old tuberculin were given, and a reading was obtained 48 hours later in 42,492 instances. An indurated area more than 5 mm. in diameter, palpable 48 hours after the injection, was taken as constituting a positive reaction. A total of 2,889 physical examinations were made, and 1,298 specimens of sputum were examined for tubercle bacilli.

Control Series

Some attempt was made to rule out non-specific allergy to old tuberculin, which might be supposed to occur in the African, prone as he is to helminth and protozoal infections. Ninety-eight hospital patients were given an intradermal injection of 0.1 ml. of 5%

glycerin broth together with the Mantoux test. Fifty-four reacted to the tuberculin, and one of these, whose Mantoux test resulted in blistering, reacted to the control solution with what appeared to be a typical tuberculin reaction. Later, 89 school-children were tested with old tuberculin and at the same time with an equivalent dose of P.P.D. (Park, Davis and Co.). Forty-eight hours later 46 showed a reaction to the old tuberculin and 50 to the P.P.D. solution.

Occult leprosy has been suggested as a possible cause of false positive tuberculin reactions (d'Arcy Hart, 1932). In a recent survey of the colony Ross Innes (1948) found an average incidence of 10 lepers per 1,000 of the population. However, his figures bear no relation to the tuberculin-sensitivity rates of the same districts. A small leper colony at Msambweni, on the coast, contains 34 cases of far-advanced leprosy. These were tuberculin tested, and 29 reactors were found, or 85.3%; a comparable age group of 909 of the general population of Msambweni gave 748, or 82.4% reactors. Of the five non-reactors, two were far-advanced lepromatous cases and three had tuberculoid leprosy.

This small control series suggests that the general results obtained are not significantly affected by non-specific reactions to tuberculin.

Tuberculin Sensitivity Rates

The results of the survey are shown in Tables I and II.

Table III shows the low sensitivity rates found by Burrows (1935) among the southern Sudanese of the Bahr-el-Ghazal province. It is here that Lyle Cummins (1939) in 1902 found no evidence of tuberculosis "among the natives, cattle, and wild game."

MacLennan's (1935) figures for Palestinian Arabs are also considerably lower than those of the Kenya survey. He tested 11,303 urban school-children of 5–13 years of age and found 1,823, or 16.2%, reacted to 1 in 1,000 old tuberculin. Of 3,660 rural school-children, 483, or 13.2%, reacted. The figures for children of the 7–12 years age group in Kenya are 37.5% and 22.4% respectively (Tables I and II, last column).

On the other hand, a recent survey of the Gold Coast by Todd (1948) showed "no virgin areas, but high rates of tuberculinization at all ages; tests with rising strengths showed nearly 100% positive by adult age." The South African Mines Report (1932, pp. 205, 206) showed that 72% of recruits, who came from various native territories, reacted to 1 in 5,000 old tuberculin on engagement at the mines, and "repeated tests with stronger dilutions would probably give 90% reactors."

In England the Prophit Survey (1948) records that "the young adult population studied shows an incidence of tuberculin sensitization of the order of 85% for males and . . . 82% to 83% for females." In Kenya the 19–25 years group of urban residence was 83.7% for men and 63.3% for women (Table I).

TABLE I.—*Tuberculin Sensitivity Rates in 3,132 African Males and 937 African Females Resident in Mombasa and Nairobi, Kenya (May and December, 1948: Mantoux 1 in 1,000). Urban Rates*

Age Group	Males			Females			Both Sexes		
	No. Tested	Reactors		No. Tested	Reactors		Total Tested	Reactors	
		No.	%		No.	%		No.	%
Under 1	28	0	0	24	1	(4·2)	52	1	(1·9)
1–3	111	7	6·0	103	8	7·8	214	15	7·0
4–6	143	20	14·0	124	20	16·1	267	40	15·0
7–12	259	109	42·1	157	47	30·0	416	156	37·5
13–18	295	190	64·4	66	30	(45·4)	361	220	60·9
19–25	705	590	83·7	188	119	63·3	893	709	79·4
26–40	1,067	954	89·4	256	202	78·9	1,323	1,156	87·3
41–	524	502	95·8	19	18	(94·8)	543	520	95·8
All ages ..	3,132	2,372	75·7	937	445	47·5	4,069	2,817	69·2

TABLE II.—*Tuberculin Sensitivity Rates in 17,078 African Males and 16,313 African Females Resident in Native Reserves, Kenya (February, 1948, to February, 1949: Mantoux 1 in 1,000). Rural Rates*

Age Group	Males			Females			Both Sexes		
	No. Tested	Reactors		No. Tested	Reactors		Total Tested	Reactors	
		No.	%		No.	%		No.	%
Under 1	424	10	2·4	466	7	1·5	890	17	1·9
1–3	1,418	73	5·2	1,383	62	4·5	2,801	135	4·8
4–6	1,527	176	11·5	1,526	215	14·1	3,053	391	12·8
7–12	3,996	874	21·9	3,155	731	23·2	7,151	1,605	22·4
13–18	3,212	1,392	43·3	2,663	1,002	37·6	5,875	2,394	40·7
19–25	1,003	706	70·4	1,981	1,022	51·6	2,984	1,728	57·8
26–40	2,638	2,149	81·5	3,258	1,951	59·9	5,896	4,100	69·6
41–	2,860	2,345	82·1	1,881	1,163	61·8	4,741	3,508	74·2
All ages ..	17,078	7,725	45·2	16,313	6,153	37·7	33,391	13,878	41·6

There is a close similarity between these results and those recorded by Wilcocks (1938, p.44), who made a somewhat similar survey of Tanganyika fifteen years ago. His many tables cannot be reproduced here. The Kenya rates are rather higher except for the younger age groups. This is also the main difference between the Kenya findings and those of Matthews (1935), who made a survey of Zanzibar. Comparison is difficult in this case because of the more complicated technique he used.

The Kenya survey demonstrates the marked difference in reactor rates between males and females and between those living in town and those in country districts. Kenya, in common with other colonies, suffers from a lack of vital statistics, and there are no figures of the tuberculosis incidence in the different parts of the colony. It does seem likely, however, that the tuberculosis attack rate is higher in the towns, or at least in the two largest, Nairobi and Mombasa, than in most country districts.

TABLE III.—*Tuberculin Sensitivity Rates (Burrows, 1935) in 2,369 Sudanese Males and 1,293 Sudanese Females Resident in Bahr-el-Ghazal Province (Old Tuberculin 1 in 500)*

Age Group	Males			Females			Both Sexes		
	No. Tested	Reactors		No. Tested	Reactors		No. Tested	Reactors	
		No.	%		No.	%		No.	%
0–5	201	18	9·0	225	14	6·0	426	32	7·5
5–10	585	125	21·4	261	41	15·7	846	166	19·6
10–25	973	367	37·7	475	156	32·8	1,448	523	36·1
Over 25	610	342	56·1	332	136	41·0	942	478	50·7
All ages ..	2,369	852	36·0	1,293	347	26·8	3,662	1,199	32·7

Men often work for a spell in the towns and return to their wives in the Reserves when they have earned a little money. When adult males were tested in the Reserves they were asked if they had ever worked in Nairobi or Mombasa: of the 5,007 men tested, 3,029 who answered "Yes" showed an 83.1% reactor rate, while those who answered "No" gave a 72.3% reactor rate.

Disease Incidence

It was not practicable to examine more than a small proportion of tuberculin reactors. An attempt was made to examine all those with chest complaints, children who reacted and their contacts, and contacts of suspected cases of the disease, and also to collect a specimen of sputum from these people. So, besides the cases missed by physical examination—which, having regard to the type of disease the African gets and the ease with which the tubercle bacillus is recovered from his sputum, may be fewer than would be expected in European practice—it is probable that some cases of tuberculosis escaped examination altogether.

It was found necessary to divide those diagnosed as tuberculous into "proved" and "suspected" categories according to the criteria already described. The number falling into the former group for all the places surveyed averaged 4.6 per 1,000. It is felt that a closer approximation of the true state of affairs would be reached if both "proved" and "suspected" cases are combined, in which case the average incidence amounts to 11.1 per 1,000. The 1948 census gives an African population of 5,252,753. If we take the incidence of only "proved" cases found during the survey as an average for the whole colony, then there are 24,000 cases of tuberculosis in Kenya. The figure of 11.1 per 1,000 gives a total of 58,000 cases of tuberculosis, and it is felt that this is more likely to approximate to the true number.

It is interesting to find that Wilcocks estimated the incidence in Tanganyika as 11.55 per 1,000. In Southern Sudan, Burrows diagnosed tuberculosis in 173 cases, which in an estimated population of 156,300 gives an incidence of 11.1 per 1,000. In Palestine, MacLennan estimated the incidence at 5 per 1,000.

It must be made clear that the Kenya estimate is based on the number of cases actually diagnosed among the general population examined in the native Reserves. No account is taken of the few cases lying in the native hospitals of the colony. Hospital figures give no indication of the incidence of the disease. Although cases of bone, joint, and glandular tuberculosis were seen fairly commonly, there is no doubt that pulmonary tuberculosis is by far the most prevalent and most important manifestation of the disease in Kenya.

As can be seen from Tables I and II, for the purpose of tabulation the sexes are divided and each sex is classified into eight age groups. In comparing the reactor rates found in different districts, it is necessary to select one age group. Adult males are often used, but, as has been shown, their reactor rate varies with their experience of the towns. Children in the 7–12 years of age group have been chosen here. Large numbers were tested. Sexual differentiation in tuberculin sensitivity rates, at least, is not marked, and males and females may for this purpose be combined. Their peripatetic habits are free but local, and when they become infected they probably do so within their own district. Table IV compares the sensitivity rates of this age group by districts. Tests which were made at various places within the same district and tribal area have been combined. The disease incidence, computed on "proved" and "suspected" cases found in the whole sample of population tested for the corresponding districts, is also shown.

TABLE IV.—*Incidence of Tuberculosis per 1,000 ("Proved" and "Suspected" Cases) in Both Sexes and at All Ages Related to Tuberculin Sensitivity in Both Sexes at Ages 7–12 Years*

District	Both Sexes: 7–12 Years		Both Sexes: All Ages
	No. Tested	% Reactors	Tuberculosis Rate (per 1,000)
	Urban Rates		
Nairobi and Mombasa ..	416	37·5	—
	Rural Rates		
Kapkatet (Kipsigis) ..	328	7·3	15·5
Mberi and Ciamberi ..	568	9·6	8·5
Kisii (S. Nyanza) ..	582	10·1	5·9
Embu	1,119	16·3	6·1
Siaya (C. Nyanza) ..	330	16·4	7·7
Maragoli (N. Nyanza) ..	188	16·6	9·5
Kajiado (Masai) ..	239	18·4	11·6
Kangundu (Machako's) ..	725	22·1	14·3
Kerugoya	697	22·5	8·9
Teita	532	29·9	8·4
Fort Hall	87	35·8	—
Taveta	105	36·2	16·0
Likoni, Kisauni, and Chamgamwe	367	38·4	8·0
Msambweni	237	39·1	9·7
Kitui	714	39·8	23·5
All rural areas ..	7,151	22·4	11·1

It will be seen that the highest percentage of reactors (39.8%) occurs in Kitui. The Kitui Reserve is dry and infertile (typical groundnut country), and the Wakamba living there are in a miserable state of poverty. They suffer from endemic malaria, schistosomiasis, and helminth infestation. They seem to be dirtier and to have a greater incidence of scabies than other Africans met with on the survey. The greatest number of "proved" and "suspected" cases (23.5 per 1,000) was diagnosed here.

After Kitui, the highest reactor rates were found at Msambweni, 35 miles south of Mombasa, and at Likoni, Kisauni, and Chamgamwe, all on the coast in the neighbourhood of Mombasa. In these places a comparatively low disease incidence rate of 9.7 and 8.0 per

1,000 respectively was found. Next come the colony's two principal towns, Nairobi and Mombasa. For various reasons, no estimate of disease incidence was possible here. Africans living in the towns are usually at work, and if they fall sick either go to hospital or, in the case of pulmonary tuberculosis, more often return to their Reserves. Thus, although it is likely that tuberculosis often develops in the urbanized African, it is not expected that a very high disease incidence would be found except among the indigenous population.

Wilcocks (1932) found that reactor rates and disease incidence in Tanganyika varied in direct proportion and that the former could be taken as a measure of the latter. This generalization is partially true of Kenya. Obvious discrepancies are the low sensitivity rates but relatively high disease incidence found at Kapkatet, Mberi, and Ciamberi—all remote and rather isolated up-country areas; while the coastal districts, as exemplified by Msambweni, Likoni, Kisauni, and Chamgamwe, show the reverse tendency with very high reactor rates and comparatively low disease incidence. It is suggested that the first phenomenon denotes a recent introduction of tuberculosis, while the second indicates a more prolonged exposure to the disease.

Type of Tuberculosis Seen in the African

Most observers are agreed that Africans suffer from a more acute and fatal type of tuberculosis than Europeans. Thus Vint (1937), in an analysis of 1,000 consecutive necropsies in Nairobi, found tuberculosis to be the cause of death in 132 cases. "The post-mortem findings are those of a low septicaemia, resembling acute miliary tuberculosis in European children"; while Wilcocks (1938, p. 52) finds "a preponderance of cases of acute galloping consumption. . . . These natives lie midway between the completely primitive races and our relatively resistant selves." Mine workers on the Rand were found to develop an acute form of pulmonary tuberculosis characterized by glandular involvement and miliary dissemination (South African Mines Report, 1932, p. 258). Post-mortem evidence from Mulago Hospital, Uganda, leads Davies (1947a) to suppose that "pulmonary tuberculosis as met with in Uganda is rarely of the 'adult' reinfection type but, in the vast majority of cases, is the result of primary infection leading to the development of a ghon focus, and this does not heal, but leads to dissemination of the infection with resulting death."

Gelfand (1944), writing of the Southern Rhodesian native, says he has never seen an African recover from pulmonary tuberculosis; and Davies (1947b) quotes Trowell as having an equally depressing experience in Uganda. Oswald (1946) had the opportunity of studying the disease in 416 Africans who developed pulmonary tuberculosis while serving in the Army in the Mediterranean area. Forty-seven cases which came to necropsy had a history of illness for an average of 5.4 months. He describes the usual destructive caseous pneumonic lesions, and found glandular enlargement in 96% of these cases. Caseation was present in 66%, and in 92% the mediastinal glands were involved.

The contrary opinion, that a chronic form of pulmonary tuberculosis is not rare in Africans, is less usually expressed, but Wilson (1928) believed it true of Kenya, and Burrows (1935) of the Sudan. Fennel (1932) writes of cases in the Butterworth area of South Africa: "Many were fairly chronic, lasting at least three years and over, showing that the natives, under their own conditions, had a considerable power of resistance." Allan (1932), who made an extensive survey of the native Reserves in South Africa, found that most of the cases with pulmonary tuberculosis were "more or less chronic."

It will be noticed that pathologists, basing their opinion on necropsy reports, are agreed on the extreme acuteness and short duration of pulmonary tuberculosis in Africans. Clinicians and field workers tend to be less pessimistic, and some believe that the consumptive African may survive some years under the conditions he is used to. The difference is to be explained by the extreme degree of selection in the material available to the pathologists. Cases of pulmonary tuberculosis are not popular in overcrowded native hospitals, facilities for treating them are not usually available, and proper accommodation is lacking. The sick African is unwilling to spend long periods in hospital, especially when no active treatment is being carried out and he feels little benefit to his health. For this reason, only those pulmonary tuberculosis subjects who are too ill to be discharged or to drag themselves from hospital are likely to die there Such cases comprise the post-mortem material of the pathologists.

During the Kenya survey a definite history was obtained from 70 of the 106 "proved" cases of pulmonary tuberculosis diagnosed. No great reliance can be placed on this, but the average duration of the disease was given as 19 months. In four cases some proof of the length of history was forthcoming, for these men had been discharged from hospitals as proved cases of pulmonary tuberculosis. Their histories were of 53, 52½, 48, and 37 months.

The very fact that infection, as demonstrated by the tuberculin test, is so widespread would seem to favour the existence of at least some chronic infectious perambulatory cases. For how could the majority of the population be infected by those acute pneumonic cases, prostrated by toxaemia and dying after a few months, which the pathologists describe?

The fact that cases of pneumonic phthisis do so badly with collapse therapy would also seem to indicate that such cases are not without exception. For Davies (1946), after 20 years' work at Kibongoto, in Tanganyika, reports an encouraging survival rate in 560 cases of pulmonary tuberculosis treated with artificial pneumothorax and ancillary measures: 42% alive after 3 years and 22% after 10.

These observations show that the fulminating type of disease, described by the pathologists as resembling progressive primary infection, is not without exception. Anyone who has had experience of tuberculosis in East Africa would agree that its usual course is much shorter and more acute than that seen in European practice.

The Kenya survey, and indeed every tuberculin-testing survey made in Africa in recent years, shows Davies's (1947a) assertion, that the African never recovers from his primary tuberculous infection, to be wrong. The majority of adult Africans have been infected and show no evidence of the disease. So far as this factor is concerned, the state of affairs is like that in Europe. However, it is probable that adult primary infection not infrequently leads to progressive tuberculosis in Africans when they are exposed to a tuberculous environment. Stott's (1950) investigation at Nakuru gaol, where tuberculosis is rife, shows that an undue mortality falls on

the small group who enter the gaol tuberculin-negative. It is likely that uninfected adult Africans, coming from their reserves to work in the towns, also are liable to develop progressive primary disease.

In view of the high tuberculin-sensitivity rates found all over the colony, it hardly seems possible to explain the extremely severe and fatal type of tuberculosis which is most commonly seen solely in terms of adult progressive primary tuberculosis, although this might in part account for the observation that tuberculosis in Africa tends to be more severe in the towns than in the reserves.

It is usually supposed that the chronic fibro-caseous type of disease commonly seen in Europe results from a partial immunity conferred by childhood infection. Many children have been found to be tuberculin-sensitive in Kenya, but only a few adults develop the chronic apical disease of Europe. It would seem that a healed primary infection may not have the same immunizing power in the African as is usually ascribed to it in the case of the European. This contention is supported by the findings of the South African Mines Report (1932, p. 97) on tuberculosis among African gold-miners on the Rand. These Africans were graded into four categories on engagement at the mines, according to their reactions to serial tuberculin tests—"positive plus," "ordinary positive," "weakly positive," and "negative" reactors. It was subsequently found that the incidence of tuberculosis was 2.9 per 1,000 for "negative" reactors, 4.5 per 1,000 for "weakly positive," 6.5 per 1,000 for "ordinary positive," and 10.3 per 1,000 for "positive plus" reactors. In South Africa Professor Lyle Cummins (1932) found that "fully 50% of healthy natives arriving at Johannesburg were capable of reacting to 1 in 1,000,000 tuberculin dilution, whereas in tests carried out by me on clinically non-tuberculous adults in Wales only 25% were found to react positively to a dilution of 1 in 100,000." In Kenya serial tuberculin-testing was not done, but of 37,460 Mantoux tests made on the general population 1,969 showed vesiculation—that is, 5.3% of the total tested and 11.8% of all reactors. In England d'Arcy Hart (1932) found less than 1% of the total tested and 2.3% of the positive results vesiculated. Wilcocks (1938, p. 31) found that in Tanganyika comparable percentages were 3.2% and 6.1%.

It does appear, therefore, that Africans are more sensitive to tuberculin than are Europeans, and it seems likely that this factor is associated with the distinctive type of disease they are liable to get.

If the same significance attaches to the tuberculin test in Africans as it does in Europeans—and there is no reason for believing otherwise—then it seems that both races are able to sustain a primary infection almost equally well. This observation is of fundamental importance, for "racial immunity" is usually thought to condition this ability. May it not be rather that racial immunity is a reflection of the power, not of sustaining the primary infection, but to form antibodies to the invader and so modify an aftercoming infection? It is not pertinent to this thesis whether the infection be endogenous or exogenous.

The African, when infected, develops a high degree of allergy to tuberculo-protein, as demonstrated by his extreme sensitivity to tuberculin, but little concomitant immunity, as exemplified by the acute infantile type of disease to which he is prone. The hyperallergic state renders him liable to an acute and destructive type of tuberculosis on reinfection, while lack of immunity deprives him of the power of confining the lesion, as in European phthisis, but leaves a "secondary" infection unmodified except by purely allergic factors, so that it often assumes the characteristics which we are accustomed to attribute to progressive primary disease in Europeans.

Summary

A description is given of a tuberculin-testing survey in Kenya.

A small control series did not show any significant tendency for non-specific factors to interfere with the results.

The sensitivity rates were found to be high in Kenya, and are compared with those of other surveys.

A greater percentage of men than women were found to be sensitive to tuberculin. Urban residence was associated with an increased sensitivity rate.

The disease incidence is thought to be about 11 cases per 1,000. If this is correct there are 58,000 cases of tuberculosis outside the hospitals in Kenya.

A degree of correlation is demonstrated between the reactor rate and the disease incidence in the various districts surveyed. Exceptions are discussed.

Reasons are given for supposing that, although the acute fulminating type of tuberculosis, resembling a progressive primary infection, is undoubtedly very common in Kenya, it is not without exception, especially in the rural areas.

An attempt is made to correlate high tuberculin allergy with the African type of pulmonary tuberculosis.

I am indebted to the Director of Medical Services, Kenya Colony, for permission to publish this précis of my report to him on a tuberculosis survey of that colony.

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During the period October 15 to November 6, 1950, 570 U.S. Navy medical reserve officers arrived at Brooke Army Medical Centre, San Antonio, Texas, for assignment to active duty with the Army. These officers were interviewed by a staff officer from the office of the Surgeon General for professional classification and assignment. They were given one day's "processing" in connexion with their entrance on active duty and received a three-day intensive orientation course before leaving for their units.

SUPPURATIVE THROMBOPHLEBITIS TREATED WITH AUREOMYCIN

BY

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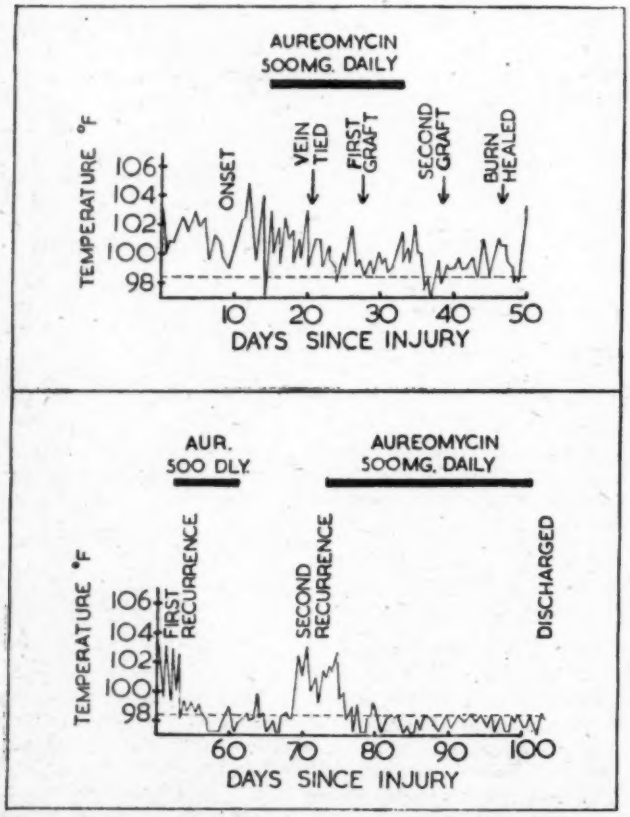
Of the various types of thrombophlebitis, the infectious and suppurative cases form only 3% (Parker, 1934). They are often fatal, however, owing to the development of pyaemia and metastatic foci. The following case of ilio-femoral thrombophlebitis is of particular interest for three reasons: (1) the prognosis was made worse by the infecting organism being a penicillin-resistant *Staphylococcus aureus*, (2) a mistake was probably made in discontinuing the chemotherapy too early, and (3) a successful recovery was nevertheless obtained with "aureomycin" without vein ligation proximal to the thrombus.

Case Report

A boy aged 3 years fell into a bath of hot water and received scalds of his back, thighs, and left arm. The extent of the scalded area was 24% of the body surface, 2% being whole-skin loss.

On admission he was treated for shock with 850 ml. of plasma during the first 27 hours. The dressing was done seven hours after admission, and the bacteriological swabs from the scalded surface at that time grew micrococci only. Systemic penicillin was begun with an initial dose of 250,000 units, and then 30,000 units four-hourly. Further swabs on the fourth day showed the presence of *Staph. aureus* and *Pseudomonas pyocyanea.*



Temperature chart during the treatment of thrombophlebitis with aureomycin.

Onset of Thrombophlebitis.—On the sixth day the child's haemoglobin was 69%, and a bottle of blood was given after cutting down on the right great saphenous vein at the ankle. Four days later his temperature rose to 105° F. (40.6° C.) (see Chart) and the pulse to 160: the right calf was grossly swollen, hot, and tender, and the drip incision was inflamed. Systemic penicillin was increased to 150,000 units six-hourly, and a course of sulphadimidine was started (2.5 g. initially, then 0.5 g. four-hourly). On the following day (13th) a small abscess was incised over the upper end of the tibia, and from the pus a pure growth of penicillin-resistant *Staph. aureus* was grown. The local inflammatory lesion subsided during the next three days, but as the temperature remained at 102° F. (38.9° C.) and the pulse at 160, aureomycin was begun on the 16th day (375 mg. initially, then 125 mg. six-hourly). On the 19th day tender, indurated, thrombosed superficial veins appeared on the medial side of the right thigh, extending to within 2 in. (5 cm.) of the junction with the femoral vein, which was not palpable. The course of sulphadimidine was stopped after a total dosage of 21.5 g., the white cell count being 5,030 per c.mm.

Operation on the Femoral Vein.—On the 21st day it was decided to tie the right saphenous vein, since the local signs suggested involvement of the superficial veins only. At operation the saphenous vein and its tributaries were collapsed and empty, but the femoral vein was found to be thrombosed, though it had been impalpable. It was opened and some of the yellow thrombus smeared on a slide and taken for culture: the external iliac vein was not explored, and was too small for an attempt at sucking out the clot. The femoral vein was tied, but the thrombus was palpable at least ½ in. (1.25 cm.) proximal to the ligature. The smear showed well-stained Gram-positive cocci, and the culture grew penicillin-resistant *Staph. aureus.*

Blood cultures taken on the day of operation and the next two days (21st, 22nd, and 23rd) were all sterile after 48 hours. An operation to tie the iliac vein above the thrombus was decided against because the child's condition was improving, the organism was known to be sensitive to aureomycin, blood cultures were negative, and there was no sign of metastatic spread. By the 24th day, eight days after aureomycin was started, his clinical condition was much better, and the *Staph. aureus* had entirely disappeared from the scald. On the 33rd day penicillin and aureomycin were stopped after total dosages of 24,160,000 units and 9,375 mg. respectively.

Skin-grafting Operations.—The granulating areas were covered with split skin grafts on the 27th and 39th days; and by the 48th day healing was complete and there was no oedema of the leg or any palpable veins.

First Recurrence of Pyrexia.—Three days later, however, the child became ill again with a temperature swinging between 99 and 103° F. (37.2 and 39.4° C.). Clinical examination and investigations revealed no cause, so that after three days a recurrence of the previous infection was presumed in spite of a sterile blood culture, and aureomycin was restarted. There was a rapid response, and the drug was discontinued after nine days (total 4,500 mg.).

Second Recurrence of Pyrexia.—After a further eight days the child's temperature again rose to 102.5° F. (39.15° C.), and again it was impossible to confirm the diagnosis. A further exacerbation of the thrombophlebitis was presumed, and a 28-day course of aureomycin was begun. The temperature became normal in a week and did not rise again. The boy was discharged at the end of this course (14.000 mg.) perfectly fit, and during the next 10 weeks showed no sign of further recurrence.

Comment

The three lines of treatment in general use for this condition are antibiotics, proximal ligation of the thrombosed vein, and anticoagulant therapy.

There is no controversy about the use of chemotherapy : the most powerful drug against the particular organism isolated should be given as soon as possible, in adequate dosage and for a sufficient period. The recurrence of the clinical picture of sepsis with too early cessation of the drug has often been noticed—for example, Hyman and Leiter, 1944. Lyons (1941) reported two consecutive cases of staphylococcal cavernous sinus thrombophlebitis treated with sulphathiazole and heparin, with recovery. Each of these cases had a bacteriaemia which lasted six and eight days respectively, and he advised continuing the sulphathiazole for three months in a dosage sufficient to maintain the blood level at 5 mg. per 100 ml. Schall (1941) reported three further cases of staphylococcal cavernous sinus thrombophlebitis successfully treated with sulphathiazole and heparin. There is little doubt that if control of the pyaemia can be obtained by chemotherapy for a sufficient time, this is likely to be successful in curing the thrombophlebitis. For staphylococcal infections the antibiotic of choice is now aureomycin (Long, 1950), and Spink and Yow (1949) have reported five cases of staphylococcal bacteriaemia treated with aureomycin, with four recoveries.

According to Van Duyn and Van Duyn (1940), the idea of trying to stop the spread of infection from a diseased vein into the general circulation originated with John Hunter in 1793, when he advised putting a compress on the vein just above the suppuration. Since then, and until the development of chemotherapy, the mortality of suppurative thrombophlebitis has been proportional to the inaccessibility of the septic focus and the difficulty of eradicating it (Rosenow and Brown, 1938). Before the advent of sulphonamides vein ligation was the only rational therapy, but the mortality was still high : septic thrombophlebitis of the neck, for example, had a mortality of 20–30% even with operation (Iglauer, 1942).

The present value of proximal ligation of the thrombosed vein is more debatable. Kern and Berman (1945) reported a case of ilio-femoral thrombophlebitis following pneumococcal lobar pneumonia, empyema, and septicaemia, which proved resistant to sulphonamides and dicoumarol ; the patient was cured, however, after ligation of the inferior vena cava above the thrombus and a course of penicillin. Which one of these procedures played the major part in the patient's recovery it was impossible to say, but they felt justified in stating that the convalescence was very definitely shortened by the surgical intervention, and the danger of embolism, minor or fatal, was eliminated. In contrast, a case described by Kirk (1947) illustrates that proximal ligation without control of the pyaemia is likely to be fatal. His case of suppurative ilio-femoral thrombophlebitis due to *Aerobacter cloacae* was temporarily controlled with streptomycin, but drug resistance developed, and when infection spread to the opposite side the inferior vena cava was tied. In spite of this the patient developed septic meningitis on the sixth postoperative day and died two days later.

In practice few would doubt the wisdom of proximal ligation when the vein was easy of access, when there was a positive blood culture, and when the organism was resistant to the drugs available. Again, there is unlikely to be disagreement in adopting a conservative attitude if the site of the proximal end of the thrombus is inaccessible, if repeated blood cultures have been negative, if the organism is sensitive to the antibiotic, and there is serious concomitant disease. Between these two extremes it may be difficult to come to a decision, but the chief indication for vein ligation is inability to control the bacteriaemia with antibiotics.

Anticoagulant therapy is of less importance, but as it may prevent extension of the thrombus it should be given when possible. An open wound such as was present in the case described is of course an absolute contraindication. It is interesting that both cases described by Lyons (1941) developed pulmonary infarcts while receiving heparin.

Summary

A case of suppurative ilio-femoral thrombophlebitis arose from a transfusion incision infected with penicillin-resistant *Staph. aureus*, and this was treated with aureomycin. At operation the diagnosis was confirmed, but because of the organism's sensitivity to aureomycin, the repeated negative blood cultures, and the sustained clinical improvement proximal ligation was not performed. Failure to give the aureomycin for a sufficient period initially is believed to have resulted in two recurrences, but after 125 mg. had been given six-hourly for 28 days (total 14,000 mg.) the condition was finally cured. The treatment of suppurative thrombophlebitis has been considered briefly.

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Medical Memoranda

Case of Ovarian Pregnancy

The occurrence of an ovarian pregnancy is still rare enough to excite interest. It is estimated to occur in 0.7–1% of all ectopic pregnancies. In recent years the subject has often been reviewed (Isbell and Bacon, 1947 ; Bolton, 1949 ; Deweese, 1949), and the numbers of authentic cases have been variously calculated as approximately 60 (Curtis, 1941), 52 (Way, 1941), and 50 (Novak, 1947).

CASE REPORT

A woman aged 21, married for one month, was admitted to hospital on October 20, 1949, complaining of pain in the abdomen. Four months before admission (three months before marriage) she first had coitus. Her next menstrual period was 14 days late but otherwise normal. The following period, which occurred 28 days later, was normal. She then married and engaged in coitus, but the menstrual period which was imminent did not occur. Seven days after her marriage she suddenly collapsed in the street and complained of dizziness, low abdominal pain, and vomiting. There was no vaginal bleeding.

She was admitted to another hospital believed to be suffering from acute food-poisoning, but was discharged after three days, though still complaining of intermittent abdominal pain, and she stayed in bed at home.

A week later a profuse vaginal haemorrhage occurred associated with abdominal pain localized to the left side. Large clots were passed, and the bleeding lasted three days.

The pain still persisted. Apart from an "interval" appendicectomy two years previously she had been quite healthy. Menstruation began at 14 and had been quite regular, with normal loss.

On admission her general condition was good; the mucous membranes were of normal colour; pulse 92, temperature 97.8° F. (36.55° C.), respirations 20, blood pressure 118/70. The abdomen was not distended, the wall moved freely, the umbilicus was not discoloured. There was slight tenderness in the left iliac fossa, but rigidity or free fluid was not detected. Examination per vaginam showed healthy vulva and urethra, softish mobile cervix. There was no enlargement of the uterus, which was anteverted, but any attempt to move it caused great pelvic pain. A tender ill-defined solid mass about 2 in. (5 cm.) in diameter was palpable in the pouch of Douglas, apparently continuous with the left adnexa. The right appendages were normal. Per speculum a healthy nulliparous cervix was seen.

The urine was normal. A blood count showed: leucocytes, 8,100; erythrocytes, 4,000,000; haemoglobin, 90%. A tentative diagnosis of a left ectopic pregnancy was made.

On the sixth day after admission a profuse vaginal haemorrhage occurred with moderate pain in the left iliac fossa. Exploratory laparotomy was then decided upon.

Operation.—This was carried out by one of us (L. G.) on October 26. On opening the abdomen the peritoneum was normal in colour, and no free blood was seen. Uterus, tubes, and right ovary were normal in appearance. The omentum was adherent to the posterior aspect of the uterus and left broad ligament by filmy adhesions which separated readily, revealing the left ovary, to which was attached a mass of blood clot the size of a tennis ball. Adjacent loops of adherent bowel were peeled off easily and the mass and about half of the ovary were removed. No corpus luteum was seen on the right ovary, nor was there macroscopic evidence of endometriosis. The abdomen was then closed in layers.

Convalescence was uneventful; the patient was ambulant on the fourth day and was discharged on the ninth post-operative day. Salpingography performed on November 26 showed patency of both tubes.

Pathological Report (J.E.).—"The specimen consists of about half of an ovary, to the free surface of which is attached a firm laminated egg-shaped mass of blood clot 4 cm. in diameter. On the cut surface of the ovary a corpus luteum of pregnancy 1.5 cm. in diameter is visible. On breaking away fragments of blood clot, villi are seen in the neighbourhood of the corpus luteum and adjacent ovarian tissue. No foetus was found. Sections show syncytial masses and chorionic villi embedded in the ovarian tissue and invading the corpus luteum. Many of the villi are already degenerated. The ovarian stroma is exceedingly vascular, but no pseudo-decidual cells are evident; the zonal layer is heavily infiltrated with polymorphs, plasma cells, and macrophages, but this reaction is not seen elsewhere in the ovary. The histological picture of the corpus luteum coincides with that of an early pregnancy, with the central core occupied by a recent organizing clot. There is no evidence of ovarian endometriosis."

We wish to thank Professor E. Farquhar Murray for his helpful suggestions and for permission to publish this case, and Mr. D. Harland for his technical assistance.

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Strangulated Spigelian Hernia

Spigelian hernia occurs along the semilunar line, usually near the level of the arcuate ligament. It penetrates the broad fascial band by which the internal oblique and transversalis muscles are inserted into the rectus sheath, but lies under the aponeurosis of the external oblique muscle. This tense aponeurosis obscures the tumour and makes it difficult to palpate. The hernia has a tough rigid neck and is often covered by a thick layer of extraperitoneal fat.

CASE REPORT

A stout woman aged 65, with a lax pendulous abdomen, had a history over several years of periodic abdominal distension and vomiting, sometimes with icterus of the skin and typical gall-bladder colic. Cholecystectomy was performed in August, 1947. The gall-bladder was small and packed with stones; the common duct was free of stones. Colic and icterus were immediately relieved, but the attacks of distension and vomiting continued unabated, and became more frequent as time passed.

On May 20, 1949, she developed sudden severe pain in the right iliac fossa. This pain travelled slowly to the epigastrium, and then became generalized. The most severe pain was constantly in the right iliac fossa. Vomiting began 12 hours after the onset of symptoms and became very frequent and voluminous. She was admitted to Mr. J. Campbell's surgical unit in the Southport Promenade Hospital 20 hours after the onset of symptoms. On admission her temperature, pulse, and respiration were normal. Dehydration was obvious and proclaimed the violence of the vomiting. Tenderness in the right iliac fossa was extreme. The patient winced when the examining hand had done little more than touch the skin. A tense oval swelling about 3 by 2 in. (7.5 by 5 cm.) was palpated in the right iliac fossa, lying within the substance of the abdominal wall, parallel with the inguinal ligament and about 2½ in. (6.25 cm.) above it.

Strangulated Spigelian hernia was diagnosed. An incision was made over the swelling and the hernia was revealed after the external oblique aponeurosis had been incised. The sac contained omentum and strangulated loops of small intestine, which were kinked and bound together by tough adhesions, suggesting a fairly prolonged tenancy in rather inadequate quarters. The neck was rigid and grasped the sac tightly; it was big enough to admit two fingers. The worst of the adhesions were divided, and the intestine was returned to the abdomen, where normal colour was rapidly regained. The layers of the abdominal wall were defined and the wound repaired. Recovery was uneventful.

COMMENT

This case is interesting, as the apparent continuation of symptoms following cholecystectomy were ascribed to some residual liver damage for which she had been treated. The onset of acute strangulation at first closely imitated acute appendicitis. The correct diagnosis was given by the development of intestinal obstruction with the presence and site of the tumour.

Reid (1949) reports a case of Spigelian hernia which imitated an acute appendicitis to perfection. The above case illustrates how deceptive this rare hernia can be and the advisability of bearing it in mind when abdominal symptoms of an obscure nature are found.

We thank Mr. John Campbell for permission to publish details of this case.

D. WATSON, M.B., Ch.B.
BETTY SCOTTER, M.B., B.S.

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Reviews

BLOOD GROUPS

Blood Groups in Man. By R. R. Race, Ph.D., M.R.C.S., and Ruth Sanger, Ph.D., B.Sc. (Pp. 290. £1 10s.) Oxford: Blackwell Scientific Publications. 1950.

Ostensibly, blood groups and clinical medicine have been living together harmoniously for some 40 years. However, the discovery some 20 years ago that there were human blood groups (M, N, and P) of negligible importance in clinical medicine was a warning sign that the ways might part sooner or later, though the discovery of the Rh groups at first suggested that after all the subjects might develop most rapidly if they worked together.

Since then great efforts have been made by both sides to pretend that their interests are really the same. This has led to great concessions by clinicians. Clinical pathologists have struggled first with CDE, then with C^w, D^u, and E^u. This book should persuade them that the time has come to abandon the chase. It is useless to pretend any longer that what is interesting and even fundamental in the field of blood groups is necessarily of the slightest interest in clinical medicine.

It may come as a shock to some clinicians to find that it is possible to write a really first-class book on blood groups without giving any definite description of a compatibility test. This omission seems entirely proper in a book which is not written primarily for clinicians.

From now on the clinical pathologist with a special interest in blood groups must recognize that he cannot aspire to more than amateur status in the subject. His daily work requires a knowledge of only small, disconnected parts of it. Acquaintance with the phenotype Le(a− b−) is no more important to him than a knowledge of the rarer poisonous fungi. On the other hand, to the geneticist each blood-group system is of approximately equal interest.

This book is written by two people who have done more than any others to advance this subject in the last few years, and therefore every worker in this field should study it closely. However, this will prove far from being a burden to anyone: the whole work is enlivened by a discreet gaiety and is the easiest possible reading.

There are very few criticisms to make. It is a little surprising to find that haemolysis by anti-Rh sera—a phenomenon very difficult to demonstrate—is mentioned twice, whereas α and β haemolysins are not mentioned at all. The authors have been a little too respectful of some published work. It is unavoidable that a good many bad papers have been published on blood groups, and if these are to be quoted the reader should be given some clear indication that the conclusions are open to doubt.

These are very minor reservations. The book will save blood-group workers untold hours of hunting through the literature. Even more important, it will make them familiar with the methods, statistical and otherwise, which Race and Sanger have used to determine the pattern of so many new blood-group systems.

P. L. MOLLISON.

WATER AND SALT DEPLETION

Water and Salt Depletion. By H. L. Marriott, C.B.E., M.D., F.R.C.P. (Pp. 80. 15s.) Oxford: Blackwell Scientific Publications. 1950.

This monograph is a reprint of Dr. Marriott's Croonian Lectures of 1946, which were first published in this *Journal*. The text has been revised to bring it up to date. He sets out, in the lucid, dogmatic fashion we have come to expect of him, the main facts of salt and water metabolism, with the causes and effects of the body's depletion of each separately and of the two combined. The diagnostic and therapeutic precepts which follow are simple and practical and have already established their value.

Dr. Marriott has clearly made valuable use of his wartime experience in India, and there are few points at which anyone will cavil. No comment is made on the view, held by many, that the urinary excretion of chloride after operation and injury bears little relation to the plasma levels, and that the Fantus test is a fallacious guide to treatment in such cases. His suggestion that salt depletion aggravates pyloric stenosis by superimposing pylorospasm is interesting and explains the unexpected improvement which sodium chloride will often bring about in these patients. His insistence on the prior claim of the oral and rectal routes for administration of fluid is salutary in these days of uncritical intravenous medication.

With this little book in their hands, the junior residents, upon whom, to Dr. Marriott's distress, the management of salt and water depletion so often devolves, will be well equipped to meet these emergencies.

R. BODLEY SCOTT.

MICROSCOPIC DIAGNOSIS OF MALARIA

Studies from the Institute for Medical Research, Federation of Malaya. No. 23. The Microscopic Diagnosis of Human Malaria. Part I. A short descriptive atlas of thick-film diagnosis. By John W. Field. Illustrated by Yap Loy Fong. (Pp. 116; illustrated. No price.) Federated Malay States: Institute for Medical Research. 1948.

In the early days of malaria research technique was practically restricted to making the wet blood film, a delicate operation not to be attempted except in the seclusion of the hospital ward. Later with increasing convenience and usefulness came the dried film, watery Romanowsky, Leishman stain, and Giemsa. But meanwhile more and more was demanded from the malaria research officer. There came the increasing use of the "thick film" with its added difficulties in interpretation, "counts" were required for certain kinds of work, diagnosis of the parasite became increasingly more important as drug treatment became more precise and specific, and in wartime examination of blood films had to be made on a very large routine scale. Many beginning such work found there were troubles and difficulties to be surmounted, and advice of the detailed and practical nature required was not easy to get.

This helpful publication is designed to meet just such needs. Dr. J. W. Field has set out in the simplest and most direct manner, and in full detail with copious illustrations, a description of practical requirements in the making and examination of blood films by the malariologist. After a brief account of the forms and appearances of the malaria parasites and points regarding parasitaemia and other matters of importance in the examination of the blood in malaria, there is a chapter on the thick film and one on interpretation of

what is shown in such films. This is followed by a description of various techniques now used by the malariologist in the making and examination of blood films, information on the various Romanowsky stains, the making of films for routine or special usage, methods of counting, practical details of Giemsa staining for different purposes, labelling of slides, and other useful practical hints. The value of the work lies not only in the very full detail given, but in the extent to which the author's own intimate experience is incorporated. The reviewer knows of no work quite comparable and has no hesitation in saying that it will be a useful work of reference and guidance not only to the beginner but to all engaged in work of the kind which the author discusses.

RICKARD CHRISTOPHERS.

ADLER'S PSYCHOLOGY

Adler's Place in Psychology. By Lewis Way. (Pp. 334. 18s.) London: George Allen and Unwin. 1950.

Alfred Adler's name is often mentioned. To the majority he is one of three—Freud, Jung, and Adler. With him are associated a few catchwords that have become part of our daily language, among them inferiority complex, will-to-power, masculine protest. The extent to which the principles of his "individual psychology" have been absorbed into current psychiatric thought is indicated only by the frequency with which they are rediscovered, with and without acknowledgment.

Mind is an abstraction, and those who would reduce its manifestations to terms of planes of space, of levels and depths, must guard against semantic confusion lest their psychogram become all symbol and no mind. It is for this reason that Adler's work has often attracted the physician who doubts the validity of the Freudian blueprint of mental life. The original works have lost a good deal in translation, and, since they have never been presented as more than sober advances in medical science, they have been read mainly by specialists. Infallibility has not been claimed for the author. Certainly Adler's principles are relevant to the present preoccupation with psychosomatic medicine and social psychiatry. Mr. Way has achieved more than a restatement of them, though he has done this faithfully. He has attempted the difficult task of finding their sources and relationships and of tracing the development of Adler's theories through the life of the man himself. As a personal friend of Adler's, and no mean student of philosophy, he is well qualified to do this. His description of the uneasy collaboration between Adler and Freud in the early days of the psycho-analytic movement, and how the attitude of each, admirable in his own way, was moulded by his own personality, makes fascinating reading. In his hands the historical approach to the tangled web of present-day theory in psychopathology brings some order into the situation. This book is no Adler Without Tears, nor is it a synopsis. It is not all easy reading, but it is a reasoned and scholarly presentation of the implications of "individual psychology" in many fields, including that of medicine, and there is here little to which the physician could take exception.

This is an important book which challenges much that is accepted by many psychiatrists. Not only will it be a valuable companion to Adler's work, but it is an admirable book for the mature physician whose experience has taught him that to know the natural history of illness he must study not only disease but man.

ALEXANDER KENNEDY.

CHEMICAL PHYSIOLOGY

Grundriss der chemischen Physiologie. By Dr. Fr. N. Schulz. Second edition. (Pp. 234. M. 7.50.) Jena: Gustav Fischer. 1950.

The author of this book on the outlines of chemical physiology treats the subject from the physiological aspect rather than the chemical. Thus he gives only an elementary account of the chemistry of carbohydrates, fats, and proteins under the heading digestion. He gives more details under intermediary metabolism, where the reader may find described (1) the scheme of the phosphorylation of glucose and its conversion to lactic acid in muscle and to alcohol and carbon dioxide by yeast, (2) the β-oxidation of fats, and (3) at some length the older knowledge of the conversion of amino-acids to amines and of tryptophan to indican. The dehydrogenation processes with various co-ferments is scantily described. Only Warburg's respiration ferment is considered. Cytochrome is not mentioned, nor is secretin under digestion, but some of Pavlov's work is discussed. The account of total metabolism is that of Voit and Pettenkofer; there is nothing about the work of Atwater and Benedict, nor the simpler ways of studying oxygen intake and carbon dioxide output. The work of physiologists outside Germany is omitted.

The best chapter is that on blood. The author regards haemophilia as due to a disturbance of thrombin and antithrombin formation. He often describes the comparative physiology in other animals, which adds some interest. The last page is devoted to a very short account of the use of radio-active isotopes. The book is disappointing, yet may be of value to German students. Our English books on biochemistry and physiology are preferable.

R. H. A. PLIMMER.

BOOKS RECEIVED

Review is not precluded by notice here of books recently received

Problem Families in Bristol. By R. C. Wofinden, M.D., D.P.H. (Pp. 60. 2s. 6d.) London: Cassell. 1950.

The National Health Service Acts, 1946 and 1949. By J. A. Scott, O.B.E., M.D., D.P.H., and others. (Pp. 1,192. 55s.) London: Eyre and Spottiswoode. 1950.

The Biochemistry of B Vitamins. By R. J. Williams and others. (Pp. 741. 80s.) London: Chapman and Hall. 1950.

Therapeutic Radiology. By G. Winslow Holmes, M.D., and M. D. Schulz, M.D. (Pp. 347. 52s. 6d.) London: Henry Kimpton. 1950.

Medical Treatment. By G. Evans, M.D., F.R.C.P. (Pp. 1,398. 105s.) London: Butterworth. 1951.

Die Funktionellen Sexualstörungen. By W. Kemper. (Pp. 102. M. 5.70 or $1.35.) Stuttgart: Georg Thieme. 1950.

Leitfaden für den Geburtshilflichen Operationskurs. By the late Professor A. Döderlein; revised by Professor G. Döderlein. (Pp. 272. M. 7.50 or $1.80.) Stuttgart: Georg Thieme. 1950.

Praktikum der Wichtigsten Infektionskrankheiten. By the late Professor C. Hegler; revised by Professor H. E. Bock. (Pp. 277. M. 14.70 or $3.50.) Stuttgart: Georg Thieme. 1950.

Amputierte und Prothesen. By Dr. R. Elle. (Pp. 38. M. 3.40.) Jena: Gustav Fischer. 1950.

Grundlagen der Gynäkologie. By Professor H. Martius. (Pp. 144. M. 23.40 or $5.55.) Stuttgart: Georg Thieme. 1950.

Atlas der Normalen Histologie und Mikroskopischen Anatomie des Menschen. By Professor E. Von Herrath and Professor S. Abramow. (Pp. 139. M. 48.) Stuttgart: Georg Thieme. 1950.

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MEDICINE IN AUSTRALIA

On January 1, 1901, the Commonwealth of Australia was inaugurated by the union of the six Colonies, which had each had a separate Government up to that time. It is fitting, therefore, that our contemporary, the *Medical Journal of Australia*, should for its first issue of 1951 publish a jubilee number which reviews the status of medicine in Australia at the beginning of the century and its progress and development in the last 50 years. The 14 papers in this special issue are not only eminently readable, but together they make a useful contribution to Australian domestic history.

During the nineteenth century the medical practitioners of Australia had come from abroad, having been trained in England, Scotland, Ireland, and, more rarely, on the Continent of Europe, but towards the end of the century medical schools had been established in Sydney, Adelaide, and Melbourne, from which already a limited number of practitioners qualified each year. In the towns the family practitioner held a position of trust similar to that of the family solicitor and banker, and his advice was asked about many matters of importance to the family, apart from illness. Consultants in the capitals of the States visited patients with the family practitioner and also saw them at their consulting-rooms. In the country conditions were different, largely because modern transport had not broken down the isolation of rural communities; it was not unusual for a doctor to ride on horseback to visit a patient some 20 miles from his home or to drive in a horse buggy. Motor-cars came in during the first decade of the new century; but the country roads were as a rule bad, and it was often impossible most of the year to approach the farms in a car.

At the present time half the population of Australia is concentrated in the State capital cities, and most of the remainder live in the more fertile coastal areas of Southern Queensland, along the borders of New South Wales and Victoria, and on the south coasts of South and Western Australia. Conditions in the big cities are much the same as in other well-populated countries, the treatment of illness being undertaken by the family practitioner with the assistance of a hospital easily available. The centres of medical work and teaching are the hospitals. These were built in the early days mainly for the destitute sick, the visiting doctors giving their services free. In the country towns hospitals were often built, as in this country, as memorials to some person in the district. During the past 50 years hospitals in Australia have grown in importance, and their function has been enlarged to include not only treatment of the sick poor but other seriously ill patients. Small private hospitals in the suburbs and country towns were established, especially for maternity and minor surgical cases, but gradually these have had to give up through being unable to meet increasing costs. New hospitals in the big centres have been built on modern lines (there are some impressive photographs of these in the Commonwealth Jubilee Number of the *Medical Journal of Australia*), and accommodation has been provided for the practice of many specialties. All this of course means that more nurses and auxiliary staff are required. The cost of building, too, has increased, for it used to be possible to build a hospital for £200 or £300 per bed, but now it may be ten times as much. The expense of providing accommodation for nurses and other staff is equally heavy, and the Government has therefore had to step in and help to build, equip, and maintain hospitals.

Naturally with the growth of population there has been an increasing demand for doctors and for a standard of medical care equal to the best obtainable in other parts of the world. This has been hastened by the impact of two world wars on Australia. Gradually medicine became a more attractive profession, and many more doctors were trained locally. Some of the best of these came to Great Britain and settled in various posts; others, having been over during the first world war and having taken a higher qualification, returned to raise the standard of practice in their own home towns. No great local discoveries were made in these early days, but no time was lost in applying in Australia the results of world-wide advances in medicine and surgery. It was during the second world war that rapid progress was made by Australian research workers, especially in solving the many problems arising in tropical warfare. New knowledge of malaria and dysentery proved of the greatest value to the medical services of the British and United States Forces. The importance of medical research in Australia has just been fittingly recognized by the award announced in the recent Birthday Honours of a knighthood to Professor F. M. Burnet, of Melbourne. The standard maintained in the medical schools of Australia has always been high. Nearly all the early professors who went to teach the basic sciences were Fellows of the Royal Society with international reputations. The stimulus these men gave to their students was carried on into their hospital training, where the teachers were, first

and foremost, practising physicians and surgeons. Shortly after the first world war postgraduate training was arranged, and this has now become a well-developed part of medical teaching; not only are there courses in medical centres for practitioners, but small groups gather together in the country towns and are given instruction by experts from the teaching centres. Professors and teachers from Great Britain and the United States go by invitation to lecture and demonstrate.

The geographical position of Australia has been responsible for some special developments. The proximity of Australia to South-east Asia, with its many diseases, necessitated strong quarantine laws, and arising from this a tropical diseases institution was founded in Townsville and continued under the control of the Commonwealth public health services. Public health services, too, have played their part in promoting maternal and child health and in lessening infectious diseases. The infant death rate has fallen from 81.1 per 1,000 births in 1907 to 25.36 in 1949; the deaths from typhoid fever in Queensland fell during 1900–48 from 29.5 to 0.1 per 100,000; and the deaths from tuberculosis have been reduced by more than 50%. The second special development has been in bringing medicine to the sparsely populated inland of Australia. Owing largely to the initiative and broadmindedness shown by the Presbyterian Church in its Inland Mission activities, the "flying-doctor" service was inaugurated, and aeroplanes now carry medical officers on visits to widely scattered stations, and flying ambulances bring patients to hospital. This service and the practicability of giving medical advice by radio through a series of linked bush stations are easing the problem of the settlement of the "out-back."

Australian medicine during the past 50 years has made much progress, contributing men and knowledge to the old world and receiving strong stimulus from the exchange of ideas, which was specially productive during the two world wars. The five medical schools now in being in the Commonwealth and the Royal Australasian Colleges of Medicine and of Surgery are inspired by those same ideals which in the past brought the profession of medicine into high honour. But the former days of individualism have passed, and there is increasing interest in the social aspects of medicine, in the prevention of disease and the promotion of health. The development of knowledge entails the increase of specialism, and the cost of the health service rises proportionately. Funds for all these needs can be provided only by the State, and this means that medical policy must come to some extent under political control. The problem of the future is to keep the status of the family practitioner in its former position of dignity and importance. There must be freedom for that individual initiative which is so characteristic of the true Australian.

COLONIAL MEDICAL SERVICE

In a recent publication of the Colonial Office the purpose of the Colonial Medical Service is defined in these words: "To deal scientifically with the host of killing and debilitating endemic diseases in the Colonies and to combat the mass ill-health of the colonial peoples."[1] There can be no quarrel with this ideal, and the nearer it comes to attainment the greater will be the international prestige of British colonial administration—a matter of increasing importance in the world to-day. The term "mass ill-health" unfortunately is no exaggeration. Elsewhere in this issue Dr. W. S. Haynes, describing the results of an extensive survey of the incidence of tuberculosis in Kenya, concludes that there are probably about 58,000 patients with tuberculosis not receiving hospital treatment in that country. Despite the gloomy view that some have taken about the chances of an African ever recovering from pulmonary tuberculosis, Haynes concludes that the evidence shows no difference between the ability of Africans and Europeans to sustain a primary infection. Also in this issue there is a description by Dr. J. C. Chartres of the medical field units which are now working in the vast "rural" areas of Nigeria. Here about 20,000,000 people have until recently been dependent on very slender medical resources, but the field units are to be a mobile medical force which will not only help in the control of major endemic and epidemic diseases but will "assess the incidence and relative importance of diseases . . . and devise measures against those which are causing most damage to the public health." The survey findings briefly mentioned by Dr. Chartres clearly prove the necessity for taking medical care to the people in this way—a conclusion also reached by B. S. Jones[2] as a result of his observations on pulmonary tuberculosis in the Nigerian African.

In all branches of medicine there is interesting and rewarding work to be done in the Colonies. The posts available in the Colonial Medical Service for medical practitioners registered in the United Kingdom number about 800, but nearly 200 of these posts are unfilled in Malaya and East and West Africa. It is generally thought—not perhaps always with justification—that this shortage of doctors is

[1] *Appointments in His Majesty's Colonial Service*, 1950, H.M.S.O., London.
[2] *British Medical Journal*, 1949, 2, 781.
[3] Report of a Meeting of the Ross Institute Industrial Advisory Committee on October 3, 1950.
[4] *British Medical Journal*, 1950, 2, 1005.
[5] Ibid., *Supplement*, 1950, 2, 137.
[6] Ibid., *Supplement*, 1949, 2, 278.
[7] Ibid., *Supplement*, 1950, 2, 30.
[8] Ibid., *Supplement*, 1950, 2, 166.

due primarily to unsatisfactory rates of pay, but an equally powerful reason for lack of recruits may be a lingering suspicion that out-of-date methods of medical administration are still to be found in some places. Professor G. Macdonald[3] has pointed out how shortage of doctors in some Colonies leads to a heavy emphasis on routine work, a consequent lessening of interest in research, which in its turn again impairs recruitment. Much was hoped of the encouragement to medical research which would come from the establishment in 1949 of the Colonial Research Service, but so far these hopes have not had much to justify them.

About three months ago the House of Lords debated the subject of medical man-power,[4] and several speakers took the view that the best way of overcoming the shortage of doctors in the Forces and Colonial Service was to enable men and women to transfer freely, without loss of pension and other rights, from one service, including of course the N.H.S., to another. The natural corollary was, as Lord Haden-Guest pointed out, that the payment received by medical officers in the three Services and in the Colonial Medical Service should be equal to that received in the National Health Service. The Government spokesman in this debate, Lord Shepherd, was obviously impressed by the arguments put forward, and told the House that he hoped something good would come from the deliberations of the interdepartmental committee which was considering the co-ordination of medical services. The thin end of what is hoped will eventually be a large wedge has already enabled doctors in the N.H.S. to transfer for a limited period to the Colonial Medical Service without loss of superannuation rights[5] (and with the grant of a gratuity at the completion of their service); and in West Africa young doctors accepting certain Colonial Office posts are thereby able to discharge their liability to military service.

For many months past the Colonies and Dependencies Committee of the B.M.A. has been negotiating with the Colonial Office "to achieve a broad equality of reward . . . not the same figures in pounds, shillings, and pence, but the same real return for services rendered in each territory, a return which is comparable with the remuneration of doctors in the National Health Service."[6] From time to time agreement has been reached on revised scales of pay in certain Colonies,[7 8] and, though much remains to be done, it is clear that the Colonial Office is as anxious as the B.M.A. that Colonial Governments will take sensible steps to attract good medical men to their service. In East and Central Africa, for instance, the salary of a medical officer in the ordinary grade used to rise from £865 to £1,320. Under a new agreement it now rises to £1,590. This may be compared with the salary of £850 rising to £1,150 awarded by the Industrial Court to assistant medical officers in the public health service in this country. Further, the income-tax payable by a single man at the beginning of the assistant-medical-officer scale in this country is about £200. In East Africa it is about £75.

In the past complaints have been made that the information given to applicants for posts in the Colonies has been inadequate, or even on occasion misleading. The Colonial Office has recently taken steps to remove this cause of dissatisfaction. There are of course still many anomalies and unsatisfactory features about conditions of service for doctors in the Colonies. The thorny problem of private practice by colonial medical officers remains unsolved. The position of the clinical specialist *vis-à-vis* the administrator may not always be happy—bad "man-management" is still sometimes responsible for much bitterness. In general, specialists are recruited from men already in the service, but a concession has recently been made to men with higher qualifications who may be thinking of joining the Colonial Medical Service, for in some Colonies they may begin their service higher up the salary scale than less well qualified entrants. Though no guarantee can be given that a young medical officer will be employed in his specialty, the Colonial Office states that directors of medical services would see that such a man was given as much work in his own subject as possible.

The attraction of medical work in the Colonies has always, of course, appealed only to relatively few of those who qualify, and this was so even in the days when remuneration was much better than that obtainable by doctors in civil practice in this country. To some life in a particular Colony may have attractions which more than counteract poor conditions of service, and to others no financial inducement could possibly overcome their dislike of living abroad. Anyone who joins a service knows fairly well the sort of disadvantages which are inescapable in even the most benign of hierarchies. It is unlikely that any single disadvantage—separation from home and often from families, the inadequacy of salaries in some Colonies, the expense of educating children at home, early retirement—would deter doctors who might be attracted to the work, but the total effect of these doubts is discouraging. However, there are signs that Colonial Governments which wish to build up medical services comparable to those in this country realize that they must provide for their medical officers conditions of service at least the equal of those obtainable in Britain, and some practical steps have already been taken to this end.

VALUE OF GASTRO-ENTEROSTOMY

The paper by Mr. D. H. Clark elsewhere in this issue raises once more the problem of relapse rate in patients who have had a gastro-enterostomy for peptic ulcer. He has studied a group of 330 patients subjected to gastro-enterostomy during the 14 years, 1924–37, all performed electively by one surgical unit at Glasgow. Nearly four-fifths were traced, a remarkable feat, and the three-dimensional graphs give an admirable representation of the results. In round figures, after 15–20 years, half the patients remain entirely free from ulcer symptoms, but in over one-third the operation had failed to prevent appreciable relapses. The majority of patients in the series had duodenal ulcer.

How do these figures compare with the medical management of peptic ulcer? From a composite curve derived from various series,[1] the corresponding relapse rate would have been reached within 3–4 years, and the medical relapse rate after 15–20 years would be of the order of 90%. Gastro-enterostomy certainly modifies the natural history of duodenal ulcer, and further statistical evidence for this well-known clinical impression comes from a comparison of Clark's series with the cases of perforation treated by Illingworth in the same city by simple suture. Both the relapse rate and the complication rate were halved in five years in those who had had a gastro-enterostomy, though admittedly the two series are not strictly comparable. In those who have had a gastro-enterostomy the load of peptic juice is divided between the duodenum and the stoma, and the threshold for the development of ulcer may no longer be reached. This division of the burden may be sufficient to arrest the ulcer-forming tendency, particularly in women, who are constitutionally less prone to ulcer, and in the elderly, and in those who have developed pyloric stenosis. Clark's series suggests that gastro-enterostomy alone still has a place in these groups.

Although the emphasis has very rightly moved to partial gastrectomy as the most certain method of cure for gastric ulcer, for duodenal ulcer many surgeons, and indeed physicians too, have a secret longing for gastro-enterostomy (preferably without vagotomy), and welcome any encouragement to undertake it in patients whose general condition is well below the average or who may be less prone to stomal ulcers. Partial gastrectomy for duodenal ulcer leads to a low incidence of jejunal ulcer, but there is a proportion of cases troubled by the dumping syndrome, and, although they may gain weight initially, most will return to their reduced pre-operative weight by the end of five years, while their sense of well-being and general nutritional state are often below par and their liability to infections is probably increased. The more extensive the gastrectomy, the greater is the risk of loss of weight and well-being. Partial gastrectomy is, however, undoubtedly the operation of choice in those who are economically endangered by their duodenal ulcer, but should be performed only when thorough medical treatment has failed.

Research is still needed for possible methods of comparison between those who do well and those who relapse quickly after gastro-enterostomy. Hermon Taylor[2] is guided by the degree of rugosity of the gastric mucosa, and reserves gastrectomy for those with exaggerated rugae, and his follow-up results will be awaited with interest. One patient out of two entirely free from symptoms 15–20 years after a gastro-enterostomy, with low mortality and freedom from nutritional disturbances, as Clark has found, should be a spur to further investigation in this field.

[1] Ivy, A. C., Grossman, M. I., and Bachrach, W. R., *Peptic Ulcer*, 1950, p. 917. Philadelphia: Blakiston.
[2] *Proc. R. Soc. Med.*, 1944, **38**, 95.

OPERATING THEATRE TECHNICIANS

Modern advances in operative surgery require complex and expensive theatre equipment and the means to maintain these precision tools in the best possible condition. Surgeons generally have not the time or inclination to give their instruments the maintenance care they require, while nurses have no training in the cutler's art and often lack the engineer's respect for precision tools. A new member of the theatre team has now become essential—the operating theatre technician—a craftsman as necessary to the operating theatre as he has long been to production engineering. For over 50 years some leading hospitals have employed men to care for the tools of surgery, to assist the surgeon at operations, and to help maintain the precise organization of a modern operating theatre. This service has expanded as its usefulness became apparent, and the number of well-trained theatre technicians has now justified the formation of the Association of Operating Theatre Technicians. The association, under its president, Sir Cecil Wakeley, has now organized a four-year course of practical training in order to safeguard the high standard of craftsmanship essential to the technician's work. Applications from suitable candidates already outnumber the vacancies available at the few training schools now open, and there is no doubt that a number of men desire to make this work a life's career.

As with all new bodies, the association has met its difficulties, due chiefly to misunderstandings of its aims and objects. The theatre technician is not the tool setter that industry knows or the instrument curator of large hospitals, nor is he the theatre orderly or theatre porter familiar in most hospitals. His aim is not to supplant any member of the theatre nursing staff, but he is a logical and intimate member of the modern operating team and joins it as a highly trained craftsman in his own right. The nursing profession should welcome this association as a help to its already strained resources, though theatre technicians will never remove the need for theatre sisters and nurses. Hospital administrators should not look upon the theatre technician as an expensive addition to their staff, for the efficient maintenance of surgical equipment quickly shows a saving in repair bills and in the purchase of "spares" essential to meet breakdowns. From the surgeon's

standpoint, he will have as part of his team a member who out-stays the ever changing theatre staffs and a craftsman who by learning each surgeon's fads and habits eliminates minor frictions and produces the right tool in good condition at the right time, thus leading to the development of a partnership of trust and efficiency essential to the quiet work of good surgery. In order to attract the right type of man the high standard of training set by the association itself must be maintained and recognized, and the association is now engaged in negotiation with the Whitley Council on this issue. Its work deserves the full support of the medical profession, for if operating theatre technicians are graded in the same category as theatre porters or orderlies the whole aim and object of the association will be lost.

QUINCENTENARY AT GLASGOW

Just 500 years ago, in January, 1451, William Turnbull, Bishop of Glasgow, founded a university, the fourth to be established in the British Isles. On that date Pope Nicholas V issued the bull granting permission for its formation and for its students to be licensed to teach anywhere in the civilized Christian world; but for many years after the foundation was extremely poor, and indeed Turnbull's original purpose was probably little more than to train clergy for his own diocese. It was only the faculty of arts that flourished, and its college began building in 1453 in the High Street near the Cathedral, at a time when Glasgow's total population was no more than 3,000 or so.

From such small beginnings, through years of obscurity, the ecclesiastical crisis of the Reformation, and the struggles between Presbyterianism and Episcopacy in the seventeenth century, the university struggled on and grew and began to flower in the Age of Enlightenment, the eighteenth century, despite the nepotism and small personal feuds that were then so rife. Glasgow was growing too, as a result of trade with the American colonies, from 14,000 in 1712 to 83,000 in 1801, and the foundation of the Royal Infirmary in 1794 provided a hospital on which to base the medical school. A professor of medicine had been elected as early as 1637, but the chair lapsed in 1646 and was re-established in 1713, to be followed in 1720 by a chair of anatomy with botany. The duties of the professors were to give lectures if five students presented themselves, and to examine candidates trained elsewhere for the M.D. degree.

It was the vigour and enthusiasm of the famous Cullen which set the medical school on its feet. After lecturing in his own private school he was brought into the university in 1746, and from this time and that of his pupil and successor Joseph Black, who discovered carbon dioxide and made Glasgow an important chemical centre, there was nearly always an annual enrolment of 100–200 medical students, among them such men as William Hunter and Smellie the obstetrician. The nineteenth century saw a further vast expansion and consolidation under such men as Thomas Graham, the physical chemist, who first studied osmosis and colloids, and Joseph Lister, who carried out his famous experiments on antisepsis while he was professor of surgery—at the suggestion of the then professor of chemistry, who knew of Pasteur's work. To-day on its 500th birthday the university which has done so much to shape Scottish life is one of the leading academic centres of Britain, and educates 7,000 students annually to play their part in scholarship and teaching, in government and law and medicine. May it in due course celebrate a successful millennium.

COMMITTEE TO STUDY GENERAL PRACTICE

The Central Health Services Council has appointed a committee to study general practice under the National Health Service. Its terms of reference are to consider and make a report on whether the existing arrangements for engaging in general practice under the National Health Service are such as to enable general medical practitioners to provide the best possible standard of service and, in particular, to advise upon (1) the range of work and standards of practice which should be expected from the general practitioner by the public and medical profession; (2) types of general practice; (3) mode of entry into general practice; (4) non-medical help; (5) equipment and environment; (6) method of remuneration; (7) liaison with hospital and specialist services; (8) liaison with local authority services.

The members are: Professor Sir Henry Cohen (Liverpool), chairman; Dr. G. O. Barber (Dunmow); Dr. S. C. Barnes (Wirral); Mr. N. F. Baylis (Clerk, Nottingham County and City Executive Council); Dr. W. Russell Brain, P.R.C.P.; Dr. J. A. Brown (Birmingham); Dr. B. Cardew (General Secretary, Medical Practitioners Union); Dr. H. Guy Dain (Birmingham); Sir Allen Daley (Medical Officer of Health and School Medical Officer, L.C.C.); Dr. E. A. Gregg (Chairman of Council of B.M.A.); Dr. W. V. Howells (Swansea); Dr. Enid Hughes (Ruthin); Alderman Mrs. V. F. King (chairman, Southampton Executive Council); Professor Hilda Lloyd, P.R.C.O.G.; Dr. W. N. Pickles (Aysgarth); Dr. A. Talbot Rogers (Bromley, Kent); Mr. E. W. Scorer (member of Sheffield Regional Hospital Board); Dr. Stephen Taylor (London); Dr. J. S. Thomas (Manor Park, London); Mr. H. P. Travis (Secretary, Bolton Hospital Management Committee); Sir Cecil Wakeley, P.R.C.S.; Dr. C. W. Walker (Cambridge). The secretary is Mr. H. N. Roffey, of the Ministry of Health.

Any person or body wishing to submit evidence to the committee should first give notice to the secretary, Room 358, Ministry of Health, Caxton House, Tothill Street, London, S.W.1.

We record with regret the death on January 3 of Sir Arthur Hall in Sheffield, at the age of 84. He was emeritus professor of medicine in the University of Sheffield, with the foundation of which in 1905 he was closely concerned.

REFRESHER COURSE FOR GENERAL PRACTITIONERS

ACUTE BRONCHITIS

BY

A. B. CHRISTIE, M.A., M.D., D.P.H., D.C.H.

Physician-Superintendent, Fazakerley Hospital, Liverpool

The English winter, "ending in July to recommence in August," affords practitioners one doubtful recompense for its ardours—namely, that all have opportunity to become closely acquainted with at least one of the common conditions they are asked to treat. Fog, damp, and cold seem especially damaging to the bronchial epithelium, and many people in fact regard an attack of bronchitis as almost a normal winter incident, from which recovery will equally normally take place in due course. Yet acute bronchitis is not always a trivial disease. In young children and infants it is often serious, carrying a considerable mortality, and in the elderly and debilitated it may well give rise to anxiety.

Pathology and Aetiology

In the earliest stage of the disease, when the cough is dry and hacking, there is hyperaemia and congestion of the mucous membrane; soon the mucous glands pour out a thick tenacious secretion which is expectorated with difficulty; later, the secretion becomes more watery and the sputum more copious and easier to cough up. These inflammatory changes may affect any part of the respiratory tract from the larynx to the bronchioles, and tracheitis, bronchitis, and bronchiolitis are pathologically one disease. The severity of the illness depends largely on what part of the tract is mainly involved and on the age of the patient. Tracheitis in the adult can produce considerable discomfort; in the infant it may produce dangerous obstruction to the respiration. At all ages bronchiolitis is serious and is scarcely distinguishable in severity from pneumonia; in the infant it may be a rapidly fatal disease.

Both climate and infection are important in the aetiology of acute bronchitis, though the extent to which either is causal cannot be determined. Bronchitis is rare in dry atmospheres, whether the climate be arctic or tropical; it is also less common in our summer than in our winter. But the connexion may not be so direct as it seems, for on foggy, damp, and cold days people tend to congregate in underventilated surroundings, and this is favourable to the spread of infection; this may well be more important than any property of the climate itself. Moreover, there may be a difference of bacteriological environment in different climatic conditions—haemolytic streptococci, for example, tend to disappear from the throats of rheumatic subjects when they go to subtropical climates and to reappear on return to a temperate zone—so that, again, what seems to be a climatic factor may in fact be largely a bacteriological one.

On the other hand, in bronchitis no uniform bacteriology has been established—*Micrococcus catarrhalis*, staphylococci, pneumococci, and streptococci, among others, may all be found. Infection seems to play an overwhelming part in infants, the baby often having very little resistance to organisms to which in later life he will become more adapted. In the adult it is possible that both factors play a part.

Clinical Manifestations in Infants

Infection of the respiratory tract in infants is always serious. The reason is probably both anatomical and immunological. The calibre of the respiratory passages is very small, so that even a moderate amount of inflammatory congestion and secretion is apt to cause grave respiratory embarrassment. Thus inflammation of the trachea is almost certain to give rise to some degree of stridor, and in many instances the condition will be one of emergency. Even when only the larger bronchial tubes are involved the diminution in respiratory volume may still be sufficient to cause symptoms which would occur only in pneumonia in the adult.

When the finer bronchioles are involved the condition is invariably desperate, and acute bronchiolitis is a not uncommon cause of sudden death in infancy. The part played by immunological factors is largely a matter of conjecture, but against at least one organism—the staphylococcus—the infant seems to have very little resistance, and deaths due to staphylococcal pneumonia or bronchiolitis are relatively common, whereas in the adult the staphylococcus only occasionally breaks completely through the body's defences. Infection usually results from exposure to an adult suffering from a relatively minor condition—for example, the common cold—and quite often the adult has no clinical symptoms at all but is a nasal or throat carrier of staphylococci or other organisms.

Acute Laryngo-Tracheo-Bronchitis

This is a new name for an old condition—"croup." The symptoms are well known, and it is not proposed to deal with them in detail. Only a few of the cases are now caused by diphtheria, but a relatively slow onset, with stridor increasing gradually over 12 to 24 hours, should still make one think of it. On the other hand, if a child awakes during the night in the grip of sudden croup the cause is unlikely to be diphtheria. Staphylococci are fairly often isolated from swabs taken from the trachea after tracheotomy, but in many cases no pathogenic organisms are found and the cause may then well be a virus.

It is always difficult to assess the prognosis, and it is therefore safer to admit most cases to hospital. A few cases will require tracheotomy, and in these delay is dangerous: the decision to operate will be based on the amount of recession of the soft parts of the chest during inspiration, the pulse, and the general condition—a child who struggles and puts up a fight may get through on its own, but the pale, listless, croupy child usually requires tracheotomy.

Most of the moderate cases get well on their own, and this makes it difficult to assess the value of any remedy. Our routine is to give a mixture containing 5 mg. of pethidine three times a day and 250,000 units of penicillin every six hours. On this regime very few

cases require tracheotomy, but whether recovery is due to the treatment or not it is difficult to say. We have also used chloramphenicol in quite a few cases, and in some dramatic improvement has been apparent. In hospitals where croup is a common cause of admission there will probably be more scepticism about the value of the drug than in hospitals where only the occasional urgent case is admitted. At the moment it is probably advisable to try the drug in all serious cases. With regard to steam treatment, the tendency is to use this less and less, but in the well-chosen "dry" case its effect can be very satisfactory.

Acute Bronchitis

This is not always an easy disease to diagnose in very young infants: apart from the fact that the baby is obviously ill, there may be very little to go on. The temperature is often raised to 102–103° F. (38.9–39.4° C.) and respiration may be noticeably increased. On the other hand, symptoms may be referred to other systems: diarrhoea and vomiting are quite often the presenting features, or restlessness and irritability may be so pronounced as to suggest the possibility of meningitis. Examination of the chest may be of little help: a few moist sounds may be heard, but noises are frequently referred from the nasopharynx. In fact, diagnosis will often be conjectural and will depend largely on excluding other causes of pyrexia. In the older infant, as the time of teething is approached, the disease tends to be less acute and the symptoms more typical of bronchial infection.

The severe case is a medical emergency and in most instances is probably better in hospital. The social background in many cases is important, but the prime cause in nearly all is infection, and it is therefore rational to use anti-infective agents. Penicillin is often effective, and can be given orally to infants under 3 months old: 50,000 to 100,000 units dissolved in a few millilitres of normal saline can be added to each feed, or in the case of a breast-fed infant can be given by spoon after the feed, either in saline or added to a little expressed breast milk; this dosage may be excessive but it guarantees therapeutic concentrations. To older infants, because of the destructive action of gastric acidity on the drug, penicillin should be given by repeated intramuscular injection; if done skilfully this is not so upsetting as might be supposed. Sulphonamides in the form of a sweetened suspension are often of value. One tablet (0.5 g.) of sulphatriad—a combination of sulphadiazine, sulphamerazine, and sulphathiazole in one tablet—can be given four-hourly to a young infant for the first 24 hours, and thereafter 0.25 g. four-hourly for the next three or four days: it is not often necessary to give the drug for a longer time. Cough mixtures of any kind are undesirable in infancy, as they may upset digestion without affecting the bronchitis.

So far as is possible the feeding of the child should not be interfered with, and if a breast-fed baby is admitted to hospital the mother ought to be admitted too; breast-feeding by a mother visiting the hospital for each feed is rarely successful unless the home is very near the hospital. If the mother cannot be admitted the infant should if possible be treated at home. The child must be kept warm and must not be disturbed unnecessarily, and bathing, except to remove soiling, should be forbidden. Many of the more severe cases will require oxygen, and the only satisfactory ways of giving it to babies are in an oxygen tent or, better still, in a "perspex" oxygen box.

The above treatment is that required for a severe case, and a large proportion of the younger cases are severe. For less acute cases careful nursing, feeding, and warmth will be sufficient.

Acute Bronchiolitis

In infants this is usually a fulminating condition, death occurring within a few hours. The diagnosis is often made only at necropsy, when the sole macroscopic finding is that thin mucopus can be expressed from some of the finer bronchioles: on section, some bronchioles will be seen to be packed with pus cells and the surrounding alveolar spaces may contain fibrin with or without some pus cells. Pathologically, the condition is, therefore, not readily distinguishable from early bronchopneumonia except that death has prevented the full development of the pathological picture. We have seen the condition develop in infants already in hospital for other conditions, but most cases arrive *in extremis* and death occurs shortly after admission. Abnormal physical signs are not usually present, and the condition can be suspected only in an infant who within a matter of hours becomes desperately ill, possibly with evidence of acute respiratory distress. Emergency treatment with oxygen and warmth is obviously required, and, if the child can be brought through the first few hours, treatment as outlined for acute bronchitis must then be energetically pursued.

Clinical Manifestations in Older Children

In toddlers and school-children there is a marked change in the aetiology and clinical manifestations of acute bronchitis. The disease loses much of its immediate severity, and death due to uncomplicated bronchitis is rare. Acute laryngo-tracheo-bronchitis still occurs, but it is less common, and in the older child the condition is less severe. There are no cases of acute bronchiolitis corresponding to the fulminating type seen in infancy; when the inflammatory condition extends, the clinical manifestations tend to be rather those of definite bronchopneumonia. On the other hand, bronchitis causes much of the minor illness of childhood and is responsible for much debility and poor health. In a proportion of cases bronchitis may represent the early stages of later chronic conditions such as bronchiectasis.

While acute bronchitis quite often occurs in perfectly healthy children, frequently it arises apparently by extension from chronic sources of infection in the upper respiratory tract. The problem of what are and what are not unhealthy tonsils and adenoids is a question one has no desire to discuss; but most practitioners have seen at least some children in whom a succession of colds and attacks of bronchitis have stopped only after the removal of the tonsils and adenoids. Whether in the successful cases the tonsils and adenoids have themselves been acting as sources of infection or whether mouth-breathing has been more responsible for the bronchitis one cannot say. Chronic nasal catarrh, often associated with enlarged adenoids, also seems to be causally related to repeated attacks of bronchitis, as does chronic infection of the sinuses. What the precipitating factors are and whether climatic conditions are as important as they appear are matters for conjecture. The bacteriology is always difficult to establish, but in some cases at least there is evidence

that one dominant pathogen which has been responsible in the past for an acute upper respiratory lesion—say, acute sinusitis—appears again and again to be responsible for succeeding attacks of acute bronchitis. Moreover, in such cases the use of a drug to which the organism is sensitive may again and again abort these attacks, but this is a line of therapy which can be successful only in individual cases.

Acute bronchitis occurs as a complication of many infectious diseases, of which measles and whooping-cough are the commonest. In all but the mildest cases bronchitis might in fact be regarded as part of the disease rather than as a complication.

The clinical manifestations of acute bronchitis in older children do not differ substantially from those seen in adults.

Bronchitis in Adults

There is no factor in the aetiology or pathology of the condition which has not already been discussed: upper respiratory foci of infection may still play a part, just as in childhood, but it is now a less obvious part. It would seem that in the adult the bronchi are, as it were, "conditioned" to the various assaults that can be made on them. Adults in this country can almost be divided into two groups—those who with distressing regularity suffer from attacks of acute bronchitis winter after winter, and those in whom bronchitis is a rarity. If properly treated the disease is not serious except in the aged or in sufferers from cardiac disease. Repeated attacks may lead to chronic bronchitis, and possibly to bronchiectasis. But, apart from these possibilities, the only danger is neglect to treat the condition early, thereby incurring the risk of pneumonia.

The onset is usually acute, with a feeling of chilliness and malaise: often mild headache and sore throat are present, and there may have been coryza for a day or two. Pain behind the sternum, accompanied by a dry, hacking, and often painful cough, is common; sputum at this stage is scanty and thick. Sometimes laryngitis with hoarseness occurs. The temperature is usually raised to 99–100° F. (37.2–37.8° C.). The patient may be a little flushed, with bright watery eyes, but there is no real distress, and many patients persist in going about at this stage in the belief that they will shake off the attack: the usual result is a severe illness. After the first day, in most cases, the sputum becomes more profuse and the cough easier. Headache and malaise disappear, and after two or three days in bed the patient begins to feel quite well, although still troubled with a cough. Most patients are up and about again within seven to ten days.

Examination of the chest will reveal nothing beyond some harshness of the breath sounds and probably diffuse moist rales. There are no areas of dullness, and vocal fremitus is not affected.

General Treatment

In most cases the illness will eventually clear up without any special treatment, but only at the cost of several weeks' ambulant illness, during which time the patients are a nuisance and danger to themselves and to other people. There is no such thing as "shaking off an attack," and there can be no doubt that the most essential thing in treatment is to confine the patient to his house and, if possible, to keep him in bed for a day or two. This measure alone will abort many of the milder cases. In the more acute cases the patient will himself take to bed. Warmth is essential, and hot bottles and hot drinks are best; it is easy to prescribe an optimum temperature for the sick-room, but in many houses it is not so easy to attain. The patient will usually adjust his own diet, and in the first day or two he will probably desire only fluids. Sedatives and hypnotics may be required; codeine phosphate, ½ to 1 gr. (32 to 65 mg.), may be given in tablet form—this has a mild hypnotic effect and will also tend to suppress the cough—but any of the common hypnotics may be used. On this regime alone patients will lose their acute symptoms within a day or two.

Treatment of Cough

The cough reflex may be stimulated by any abnormal condition of the bronchial tract: afferent sensory impulses are conveyed from the area of irritation, possibly via the sensory fibres of the vagus nerve, to the cough centre in the medulla, where the motor side of the cough mechanism is controlled. In acute bronchitis three factors may play a part: (1) spasm of the bronchial musculature; (2) inflammation and congestion of the mucous membranes setting up afferent impulses as a result of irritation of sensory-nerve endings; and (3) mucus or mucopus lying on the walls or in the lumen of the bronchi and acting as a foreign body. We must consider whether we have any remedies for these three factors.

Spasm of the Bronchial Musculature

The parasympathetic nerve supply to the muscles, through the vagus, causes bronchial constriction, and anything which antagonizes the action of the parasympathetic system should produce relaxation. Belladonna and atropine both have this action and when spasm is a prominent feature they may bring great relief. A very useful pill is one containing extractum belladonnae viride, ¼ gr. (16 mg.), and codeine phosphate, ½ gr. (32 mg.); this preparation is also excellent when a dry distressing "throat" cough causes sleeplessness. Stimulation of the sympathetic also causes relaxation of bronchial muscles, and ephedrine, ¼ gr. (16 mg.), three times a day may be used instead of atropine or belladonna.

Inflammation of Mucous Membranes

In every case inflammation is present; and in many cases the most prominent feature is substernal soreness with a dry distressing cough and little sputum. The traditional prescription of a stimulant expectorant in order to promote secretion and so, as it were, lubricate and soothe the inflamed area is of doubtful use. Much more effective is the inhalation of steam, medicated with tincture of benzoin. The moist warmth tends to allay the irritation and probably promotes secretion more effectively than any expectorant. In young children the vapour can be administered only by some form of steam-kettle; older children and adults can inhale from an orthodox inhaler or from a jam-jar. When these methods are not successful, codeine either as a tablet or in syrup of codeine should be given.

Mucus in the Bronchi

The use of an expectorant or cough mixture to influence the secretion of the bronchial glands is very much hallowed by tradition, and a wide, or even wild, variety of drugs has been used. These drugs are said to act in one of three ways. Those in the first group act as gastric irritants—for example, ipecacuanha, ammonium carbonate. In large doses these drugs cause vomiting, and with the vomiting there is often copious expectoration, the bronchial mucous glands being reflexly stimulated through the vagus centre: their use as expectorants is based on the assumption that sub-emetic doses

will still excite secretion in the bronchial mucous glands. The second type of expectorant is exemplified by potassium iodide. This drug is excreted partially by the bronchial mucous glands and in passing through them stimulates secretion. This certainly occurs in patients hypersensitive to iodide, but whether there is any such action in normal individuals is doubtful. Drugs in the third group of expectorants, of which tolu and benzoin are examples, are excreted by the bronchial mucous membrane, where by irritation they are said to excite secretion; drugs in this group also act as gastric irritants.

There is a tendency to divide expectorants into two groups —stimulant and sedative expectorants—although the only possible pharmacological effect of an expectorant is to promote secretion. At least three different interpretations of the term "stimulant expectorant" can be found in the literature: most commonly it means that the expectorant is one which induces a copious flow of secretion in the early "dry" stage of inflammation; it may also be applied to expectorants such as squill and ammonium carbonate, which have a secondary stimulating effect on the heart; and, thirdly, it is used to indicate that certain expectorants apparently stimulate repair of the bronchial mucous membrane. The term "sedative" is usually applied to a cough-mixture rather than to a single expectorant drug and indicates that the mixture contains both an expectorant to promote flow of secretion and a drug such as codeine or heroin to allay the patient's cough—a combination which does not sound completely logical. When the word "sedative" is applied to the expectorant drug itself it has no clear meaning, unless it be that the drug, in being excreted through the bronchial mucous membrane, at once stimulates the glands into activity and soothes the mucous membrane by some other property: but is there any evidence of such activity?

Often both stimulating and sedative expectorants are combined in the same mixture reminding one, perhaps not unjustly, of the tragi-comical classification of Polonius. Is there any real justification for the use of expectorants? Is it really justifiable to flood the system with gastric irritants in order to treat a cough? It will be argued that cough-mixtures are often effective: this is true, but is their action due to anything more than the sedative effect of chlorodyne, codeine, or heroin?

Secretion from the bronchial glands can be stimulated effectively by simpler means. Copious hot fluids by mouth will almost certainly be effective, with or without the addition of alkalis: the fluid and the heat are probably the essential constituents in the many recipes for gruel and also the varieties of alcoholic hot drinks. Inhalation of steam will also aid secretion, often very rapidly. It is doubtful if any combination of expectorant drugs can produce an effect comparable with that obtained by these two simple measures.

In many cases the mucous glands secrete abundantly, and the problem is to reduce secretion. The parasympathetic system controls secretion from the glands, so that atropine and belladonna, by antagonizing the parasympathetic, will tend to reduce secretion.

In an attempt to allay cough, then, we have seen that atropine or belladonna will allay spasm and dry up secretions if excessive: a painful, dry hacking cough can be soothed by steam inhalations, and these, along with copious hot drinks, will also help to promote secretion if this is thought necessary. In many cases cough can be relieved along these lines, but when it is specially persistent or distressing codeine should be given. Syrupus codeinae phosphatis *B.P.C.* contains ¼ gr. (16 mg.) of codeine phosphate in 1 dr. (4 ml.), and to an adult 2 dr. (8 ml.) three times a day for several days is not excessive—the *B.P.* dose of codeine is ½ to 1 gr. (11 to 65 mg.). Small doses are not effective. There is the theoretical objection that cough should not be repressed when there are secretions to be coughed up, but in a disease such as acute bronchitis there is no danger, except in very debilitated patients and young children. Linctus codeinae *B.P.C.* contains ¼ gr. (8 mg.) of codeine phosphate in 1 dr. (4 ml.). It is really the syrup of codeine diluted with the syrups of wild cherry and tolu, and there is no advantage in its use. The pill already mentioned, containing codeine phosphate, ½ gr. (32 mg.), and extract of belladonna, ¼ gr. (16 mg.), brings great relief.

Specific Therapy

It has been pointed out that a variety of organisms may be isolated from cases of acute bronchitis—pneumococci, streptococci, *Micrococcus catarrhalis*, and so on —and it cannot be claimed that any are responsible for the condition. Moreover, in a disease in which acute symptoms tend to subside so quickly there is little chance of bacteriological investigation, so that any treatment aimed at a causal organism must be a chance affair. Nevertheless the use of sulphonamides or penicillin often seems to produce good results, although it must be emphasized that improvement may be due to the general treatment of the patient. In children of school age who suffer from recurrent attacks sulphonamides do sometimes abort the attack if given at the onset; their repeated use requires caution.

Penicillin is best given by inhalation if possible: an electric atomizer is the ideal method, but there are available several small plastic inhalers which are satisfactory; these are usually charged with 100,000 units of penicillin, as powder in some and as liquid in others, and this treatment is carried out every three or four hours, one charge being enough for three or four treatments. If injections are given, then the best preparation is a mixture of plain penicillin and procaine penicillin, and injections need be given only once every 12 hours. There is no indication for any of the newer antibiotics, and it must be emphasized that the use of any anti-infective agent is entirely empirical. In most cases confinement to bed and simple methods of treatment will prove effective.

MEDICAL FIELD UNITS, NIGERIA: THEIR FUNCTIONS AND WORK

BY

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This article may not contribute any new knowledge to medicine, but it is intended to give a picture of a relatively new field in the work of the Colonial Medical Service. In Nigeria a system of hospitals, dispensaries, and their ancillary services has been established and is being expanded in the larger cities and towns, and a considerable amount of preventive work has also been accomplished, again largely in the cities and towns. In the rural areas, which are vast and have a population of some 20,000,000, much remains to be done. Until recently the staff available consisted of Government or native administration dispensary attendants and sanitary inspectors, who were given what supervision was possible by a medical officer already more or less fully occupied with the curative and preventive work at his headquarters station. The sole exception was the

Sleeping Sickness Service—a Government service fully described by Lester (1938), Harding (1940), and McLetchie (1948)—which, by a programme of mass surveys, repeat surveys, dispensary treatment, chemoprophylaxis, and anti-tsetse clearance, has in the last 20 years brought human trypanosomiasis under control. This service, although it undertook a variety of duties in rural areas, confined its activities to the endemic sleeping-sickness areas in the central northern provinces.

In 1946, as part of the 10-year development plan for Nigeria, a new rural service was formed to deal with major endemic and epidemic diseases on similar lines. Originally called the "epidemic teams," the new service is now known as the Medical Field Units, and is administered jointly with the older Sleeping Sickness Service. The intention was to have 18 provincial units, one for each of the larger provinces. Each field unit should have a medical officer, a superintendent (ex-R.A.M.C.), 18 to 24 dressers, and drivers, clerks, messengers, and other junior staff.

At present eight units have been trained and six are at work. The field of operations covers almost every type of country and climate of tropical Africa. In the extreme north, the Sokoto and Bornu units may work in semi-desert conditions; parts of Bornu still have excellent shooting. The Plateau Province unit operates partly on the tin-bearing high plateau with its rocky outcrops, and partly beyond the escarpments in the foothills, where sleeping sickness and resettlement schemes are major problems. In the so-called "Middle Belt," farther south, the Benue unit works in undulating well-watered orchard country; its work is described below. To the west a unit has been established in Abeokuta Province, north of Lagos, and to the east the Cameroons unit alternates between the coastal rain forests and the tsetse-free highlands of Bamenda, where fine herds of cattle are pastured. The two new units will go to Warri and to Rivers Provinces in the Niger delta to lead an amphibious life.

A primary duty of the units is, as their original title suggested, to assist local staff in controlling epidemics. Since 1946 they have been called on to deal with yellow fever, relapsing fever, smallpox, and cerebrospinal fever. In 1949 and 1950 a total of 100,000 cases of cerebrospinal fever were reported, and in the former year the Benue unit was transferred a distance of 800 miles to assist in the emergency. Transport is mainly in the medical officer's car, with lorries and kit-car for staff and equipment, but may be by rail, river, cycle, horse, or on foot. The last method, if an early start is made and the trek completed by moonlight or in early morning, is probably the most enjoyable for those who find delight in the countryside, which abounds in interest to the nature lover.

The other principal function of the units is to assess the incidence and relative importance of diseases in the rural population and to devise measures against those which are causing most damage to the public health. As in most parts even of the civilized world, our knowledge of disease incidence is faulty, and is based mainly on hospital in-patient and out-patient attendances. True incidence can be gauged only by examination of the whole population or of an adequate sample; such a survey is described here. This information is of first importance to the economics of a country 95% of whose population are peasant farmers producing groundnuts, palm oil, cocoa, beniseed, etc., on which the wealth of the country depends.

Survey Organization

This is best described by outlining the background of a survey done by the Benue unit during the 1950 dry season. The administrative district of Chinkai was chosen by consultation with local administrative officers. The aims of the survey were to examine quickly for sleeping sickness, scabies, ulcers, yaws, and other easily diagnosed conditions every man, woman, and child in the district, and, at the same time, to make a more detailed examination of about 300 people living in two hamlets near Chinkai town. In obtaining attendances a variety of local native administration officials assisted—district head, Emir's representative, tax census scribe, N.A. policemen, and village and hamlet heads. No previous full survey had been made, but surveys for sleeping sickness had been done. Attendance for sleeping sickness examination is obligatory, but for other work tact and persuasion have to be great to ensure full voluntary co-operation.

Chinkai district occupies a triangle of land bordered on the north by the Benue River, the main tributary of the Niger. Chinkai town is on the river at least three days' trek from divisional headquarters at Wukari. The obvious supply route was by river in canoes and poling barge, downstream some 30 miles from Ibi, the "port" for Wukari. The unit worked in two sections. "A" section had been trained by the medical officer in the technique of detailed surveys; "B" section, under the superintendent, did general surveys.

As usual there was no building suitable for survey work, and a temporary examination shelter of grass mats and poles was erected to the units' standard pattern (Fig. 1). For dry-season work a flimsy flat roof is sufficient; during the rains the roof is gabled and thatched. At the entrance, people assemble household by household and hamlet by hamlet according to their tribal groupings, and when called by the census scribe enter waiting shade-shelter No. 1, which has a shade roof only and no walls. The unit's scribe then records in his register each person by name, household, age, and sex, and for women of child-bearing age data such as number of pregnancies, number of children alive, etc. The medical officer then takes over. He examines each person for signs of vitamin deficiency, trypanosomiasis, yaws, scabies, leprosy, ulcers, eye diseases, evidence of previous vaccination, etc. He gives each a card with directions for laboratory investigations and treatment, if required. Purdah women, of whom there were a few at Chinkai, the wives of the more prosperous Moslem traders, were examined in privacy behind a spiralled mat screen; all adult males were examined for venereal disease behind a similar screen. After seeing the medical officer the people wait in a second waiting-room, where vaccination and revaccination are done.

The second waiting-room leads to the largest shelter, over 40 ft. (12.2 metres) long. This is divided into laboratory and treatment sections, the latter subdivided into a "clean" injection section and a "dirty" dressing and general section. Laboratory investigations are necessarily restricted to what is practicable under field conditions. Dust, heat, and flies can prove trying to the temper, to aseptic technique, and to the preparation of specimens.

At a detailed survey the routine investigations made on all attending are: wet blood film, 10 minutes; stained thick film (Field's or Giemsa), 10 minutes; stool; urine—tested for albuminuria; if positive the specimen is

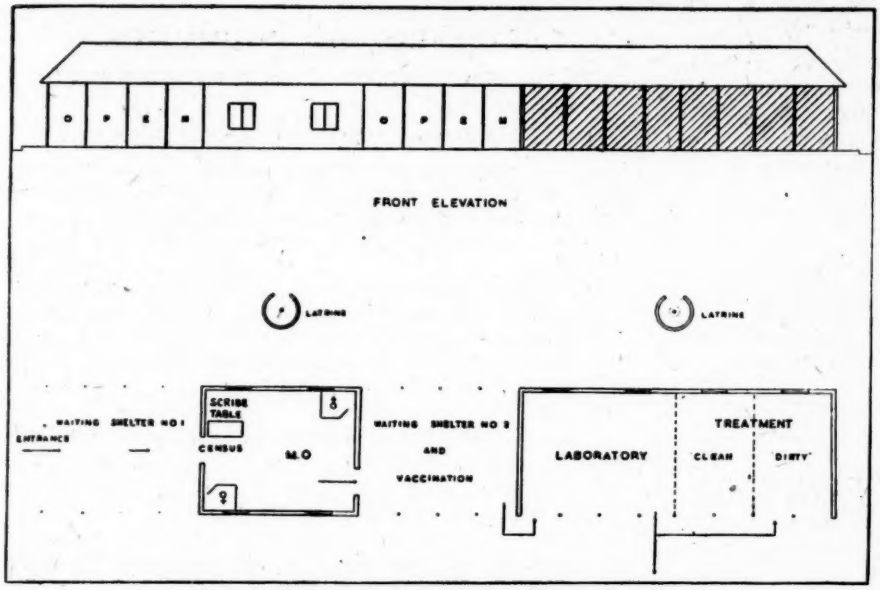

FIG. 1.—Lay-out of temporary examination shelter.

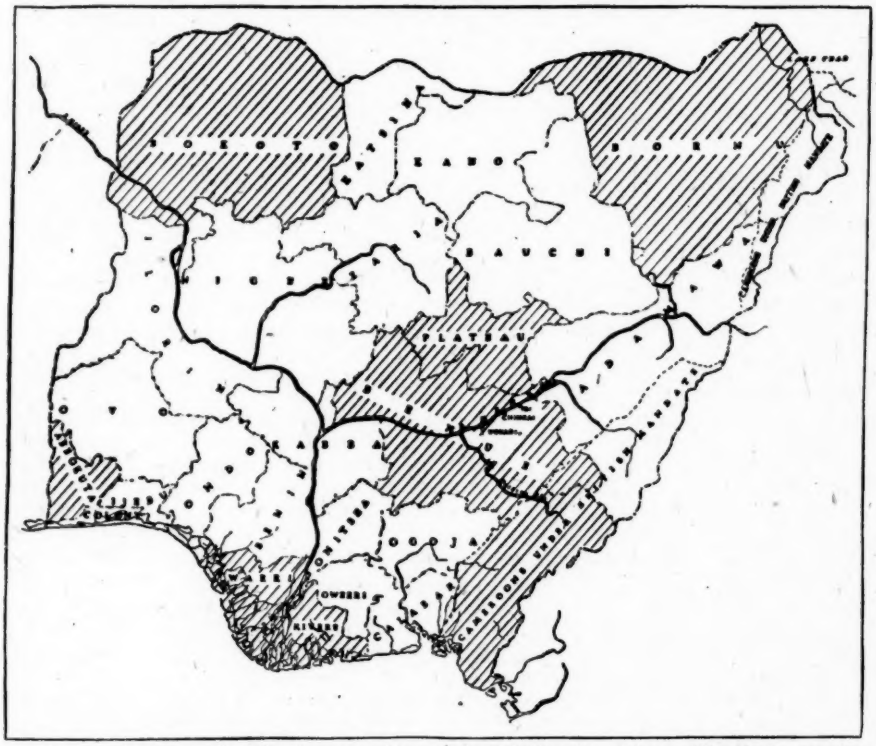

FIG. 2.—Provinces in Nigeria covered by medical field units. *Note.*—Nigeria in area is greater than France, Italy, and Belgium combined; in population greater than populations of Canada, Australia, and New Zealand combined.

centrifuged and examined for red cells and schistosome ova; haemoglobin—the Sahli method is now replacing the Talquist.

In addition, in those with suspected glands, skin lesions, signs, or symptoms, gland juice is examined for trypanosomes, skin snips and nasal smears for leprosy, sputum for tuberculosis, and urethral smears for gonococci. Red and white cell counts, erythrocyte sedimentation rates, Ide tests, and examination of cerebrospinal fluid may also be done. On a busy day six microscopes may be manned continuously.

In the therapy section standardized treatment is given for trypanosomiasis, yaws, schistosomiasis, scabies, etc., and a good deal of general medical work is done. Dental extraction is a popular sideline, and one soon realizes what a fallacy it is that the African has good teeth. Apart from the "surveyees" for the day, there is usually an out-patient clinic of "strangers," mostly canoemen and traders, and of villagers from areas not yet surveyed. The cases seen at these out-patient gatherings are most varied and interesting.

At the back of the clearing in which stands the examination shelters is a row of in-patient huts, where the seriously ill are treated. Some are on sulphonamides for meningitis or pneumonia—the latter being surprisingly common in the cold dry season—others on penicillin for osteomyelitis; while at Chinkai we had a series of cases of acute amoebic dysentery which required emetine under supervision. One of the huts is generally reserved for minor operations, performed on a tubular steel stretcher raised on two boxes; this makes quite a useful operating table.

At the conclusion of a survey two reliable dressers remain behind to complete the courses of treatment. These are made as short as possible to reduce absenteeism. The intensive one-day "neo-halarsine" or "mapharside" treatment of yaws is efficient and non-toxic; trial is being made of "miracil-D" in schistosomiasis; daily pentamidine is replacing suramin ("antrypol") in trypanosomiasis, although all cases still get tryparsamide; scabies is effectively dealt with by "tetmosol" in palm oil.

Before starting a survey a varying amount of propaganda and explanation is required. The medical officer or superintendent makes a preliminary propaganda visit to meet village heads and elders. During the survey specimens of vectors, such as tsetse fly, lice, and snails, and magnified coloured pictures of trypanosomes, itch mites, filaria, etc., are shown and explained; chiefs and elders are shown the larger parasites, filaria, and schistosome miracidia under the microscope; and a good deal of simple health propaganda is done. In the near future a section of the unit will be stationed in a special development area after an intensive survey, and an attempt will be made to control the more common and deadly endemic diseases in this limited area.

The Chinkai Survey Findings

At the special survey done by the medical officer, 271 people from a Jukun fishing village and from a Tiv farming community, two distinct cultures and tribes, were examined. The diseases in which they showed a possibly significant difference in incidence were:

Disease	Jukuns %	Tiv %
Trypanosomiasis	2	4·7
Malaria, spleen rate	57	29
,, average enlarged spleen	1·75	1·5
Yaws	8	16
Gonorrhoea, adult males	—	7
Schistosomiasis	1·1	39
Ankylostomiasis	4·3	29
Filariasis	6·4	29·5
Avitaminosis	Low	Moderate
Anaemia	22	8·6

Other conditions noted were leprosy (0.5%), skin diseases (2.5%), ascariasis (2.6%), previous smallpox (14.4%), and goitre (0.5%).

At the general survey of the district 3,638 people were examined. Figures which are reasonably comparable because of their straightforward diagnosis are:

	Special Survey (271) %	Chinkai District (3,638) %
Trypanosomiasis	3·4	1·7
Yaws	12·3	8·8
Gonorrhoea, adult males	4·0	3·2
Ulcers	Nil	0·3
Skin diseases	2·5	3·8
Eye diseases	Nil	1·7
Previous smallpox	14·4	11·9
,, vaccination	11·9	6·9

In 1937, 13% of the population of Chinkai had trypanosomiasis. By 1944 the incidence had been reduced to 4.4% and is now 1.7%. The principal public health problems throughout the district are malaria, trypanosomiasis, leprosy, yaws, venereal diseases, and smallpox, with schistosomiasis probably a local problem.

Summary

The functions and the work of the Nigerian Medical Field Units are described, and some of the findings of a field survey of Chinkai District, in Benue Province, are given.

This paper is published by permission of the Director of Medical Services, Nigeria. I wish to acknowledge the help received from Dr. J. L. McLetchie, senior medical officer, and, in the field, from Mr. J. G. Messenbird, field units superintendent, and the staff of the Benue Field Unit.

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Dr. J. Greenwood Wilson, medical officer of health for Cardiff, writing in the December 29 issue of the *Municipal Journal*, comments on the cautious start made with B.C.G. vaccination in Britain. Only selected tuberculosis contacts are inoculated, and every child so treated must have been Mantoux tested, and must be away from all risk of tuberculous infection for the six weeks before inoculation and the six weeks after. For this reason local authorities have been very busy staffing and opening the necessary residential hostels where the children can be kept for twelve weeks. But, although the B.C.G. scheme is their responsibility to plan and operate, the vaccine is kept and distributed by the hospital boards, and almost any medical man can obtain some and give it, without even being obliged to keep any records. He believes it would be much better if B.C.G. were made wholly the responsibility of one body to administer and record, as is at present the case with immunization against diphtheria.

Reports of Societies

LIVERPOOL MEDICAL INSTITUTION

At an ordinary meeting of the Liverpool Medical Institution held on November 30, 1950, with the president, Dr. DAVID JOHNSTON, in the chair, Mr. R. MARCUS read a paper on "Tracer and Therapeutic Studies with Radio-isotopes."

Mr. MARCUS discussed the application of nuclear physics in medical and biological research. He described tracer and therapeutic studies done with Professor Rotblat and in collaboration with the staff of the Radium Institute and clinicians in Liverpool.

Radio-isotopes in Malignant Disease

The first application was to find a reliable test for doubtful superficial carcinomata of the breast. Diagnostic experiments were made after injecting 100 microcuries of ^{32}P intravenously. The method consisted in measuring simultaneously the activity over the carcinomatous breast and of a corresponding area in the normal breast by means of two Geiger-Müller counters. In most of the cases investigated the uptake over the carcinoma was found to be much faster and followed by a faster rate of decay than on the normal side. It was established, however, that this type of uptake was not specific for carcinoma and was in some cases due to increased vascularity.

The second application was in the study of multiple melanomatosis. The results showed that radioactive copper (^{64}Cu) and di-iodotyrosine labelled with ^{131}I were not preferentially taken up by the tumours. But radioactive phosphorus (^{32}P) was taken up in sufficient amounts to justify treatment with this isotope. A case treated with 20 millicuries of ^{32}P was reported. Remarkably few changes were noted in the blood, liver, kidneys, and bone marrow which could be directly attributed to internal radiation.

Radioactive Iodine and Thyrotoxicosis

The third application was in the diagnosis of doubtful cases of thyrotoxicosis, especially in cases following partial thyroidectomy. Out of 40 cases studied 21 fell into this category. It was possible in all but one case to establish a diagnosis, which was confirmed by the results of therapy or further observations made on the patient. The method adopted was to estimate the uptake in the thyroid, the urinary excretion, and changes in the ^{131}I content of the blood. An attempt was made to devise a simple method which would give a reliable result within an hour. So far this had not been accomplished, but the investigations were proceeding. Nor had it proved possible to confirm the reliability of easy methods published recently from various other sources.

^{131}I was shown to be of value in locating the position and size of functioning thyroid tissue in abnormal positions. In the post-operative cases investigated, recurrences were shown to occur in the pyramidal lobe, lateral lobes, in front and behind the trachea, and in the mediastinum.

Three cases of thyrotoxicosis each treated with 2 millicuries of ^{131}I were investigated. The first patient had recurrent thyrotoxicosis many years after thyroidectomy. Two months after treatment, tracer experiments were repeated. The thyroid tissue had shrunk and the urinary excretion had increased, showing that the thyroid function had been beneficially affected. The findings corresponded to the clinical improvement. The second patient had a toxic adenoma which caused cardiac failure. She did not tolerate thiouracil. ^{131}I was given pre-operatively with good effect. The third patient also had cardiac failure due to thyrotoxicosis. She too had benefited from treatment with ^{131}I, although it was too early to judge if the effect would be permanent.

Correspondence

Knightsbridge Plague Hospital

SIR,—At the moment of writing contractors are busy with excavations and stonework at Knightsbridge Green, halfway between Harrod's stores and the top of Sloane Street. A notice exhibited on one of the trees intimates that a garden is being constructed there for the benefit of Londoners. This small triangular open space is exactly opposite the establishment of Messrs. Tattersall, the mart for horse sales. The tradition connected with it is that on the site of "Tattersall's" there existed in ancient times a lazar house, or leper hospital. At the time of the Plague of London, in King Charles II's reign, this leper hospital, then well outside London, was either wholly or partly empty owing to the fact that leprosy was a disappearing disease in England. It was accordingly made use of as an emergency hospital for plague cases from London. Most of the patients died, and their corpses were disposed of by digging a huge pit outside the front door and shovelling them in with quicklime. When the plague died out it was ordained that this burial pit should remain for ever undisturbed for fear of starting off the epidemic again; and so it has remained unbuilt upon to this present day. After more than 280 years it is improbable that any *B. pestis* remain in the soil, though conceivably there may be rats in Messrs. Tattersall's premises by whom the infection might be spread, if there were any. The inhabitants of Knightsbridge can be assured, therefore, that they run no risk from the disturbance of this once plague-inseminated soil; in any case the shallow diggings do not seem to have reached to the stratum where the bones may be presumed to exist.—I am, etc.,

Tunbridge Wells. HENRY ROBINSON.

Exercises in the Bath

SIR,—I regret that Sir Leonard Hill's letter (December 30, 1950, p. 1494) does not enable me to reconcile my opinion of the energy value of exercise with the statements in his article (November 18, 1950, p. 1153) to which I took exception. But since he expects me to study the pamphlet of Mr. Togna, the originator of the exercises, I feel it only right to observe that I would not have ventured to criticize an eminent physiologist without preliminary careful consideration of the subject at issue. I was one of the very first to whom this pamphlet was submitted, and Mr. Togna himself demonstrated his exercises to me eight years ago.—I am, etc.,

London, W.1. ADOLPHE ABRAHAMS.

Atypical Pneumonia

SIR,—In the articles (December 30, 1950, pp. 1457–63) by Professors C. H. Stuart-Harris and S. P. Bedson, no mention seems to have been made of the fact that some sufferers from atypical pneumonia, though non-syphilitic, give positive syphilitic serum reactions. One would have thought the fact worth mentioning, because every now and then a recent sufferer from this respiratory disease happens to have his (or her) blood tested for syphilis, and the development or not of pecks of worry for the sufferer depends on whether the medical attendants in the case know this cause of non-specific reactions to syphilitic serum tests. Diffidently I suggest also that the syphilitic serum tests might be a means of differentiating some of the group of conditions now classed as atypical pneumonia. I have butted in here because the description by Professor Stuart-Harris seems rather closely similar to some cases quoted by Osmond and myself in a paper read before the Medical Society for the Study of Venereal Diseases in June, 1943, entitled "The Uses and the Limitations of the Serum Tests for Syphilis." Referring to atypical pneumonia in this paper, we said:

"But perhaps more misleading than the conditions already mentioned is a transient bronchopneumonia which apparently gives rise to very little constitutional disturbance and may cause a positive reaction persisting for two months or longer. Particular attention was drawn to this condition by Fanconi (1936), who reported positive reactions in three undernourished schoolchildren, and one convalescent from measles, who were suffering from what was described as "hilifugal bronchopneumonia" in which radiographs showed a streaky infiltration of the lung. The serum reactions persisted until the radiographic appearances had become normal—that is, for a number of weeks. Fanconi quoted Forssman (1932) as having found only five false positives in 7,711 general clinic cases, but three in 633 cases of pneumonia, and Pockels (1933) found four false positives in 206 convalescent measles cases. Fanconi's observations were later confirmed by Hegglin and Grumbach (1941) and by Jahnel (1941). Hegglin and Grumbach had 18 cases of atypical, almost afebrile, pneumonia with the streaky infiltration mentioned by Fanconi and positive syphilitic serum reactions, and Jahnel reported 11 similar cases. Both his and Hegglin and Grumbach's cases had a high proportion of Pfeiffer's influenza bacilli in the sputum, and their sera strongly agglutinated this organism. It seems possible that Pfeiffer's bacilli may be capable of provoking pseudo-syphilitic serum reactions in patients with labile serum."

Both Fanconi's and Hegglin and Grumbach's articles are illustrated by radiographs.—I am, etc.,

London, S.W.1. L. W. HARRISON.

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Treatment of Lumbar Disk Lesions

SIR,—We must all be grateful to Dr. James Cyriax (December 23, 1950, p. 1434) for bringing some sort of light and order to clarify our ideas about lumbar disk lesions. I hope his remarks on the importance of lumbar lordosis will be widely noted and followed. Here I just want to mention one way in which they are, at present, often flagrantly flouted.

I do not now frequent large hospitals, but I find that surgeons still have a habit of directing that where a patient is being anaesthetized for a laparotomy the hands should be placed underneath the buttocks. This reverses the normal lumbar curve, under the anaesthetic the spinal muscles relax, and so the lumbar spine goes into marked flexion. This is, I believe, the reason that numbers of patients after operation—and especially when they get home, where they speak more freely than in hospital—complain of backache more than anything else. It is of course still more common after the lithotomy position.

Physiologically I think that the right place for the arms is across the chest, or if this gets in the way of the surgeon they should be crossed in the operation gown and allowed or made to drop back towards the sides in such a way that the gown exerts pressure on the thorax. In practice this is easy to arrange. Surgeons seem invariably to say that this will impede breathing, but the fact is that inspiration is caused by active muscular action, which can easily lift the weight of the arms, while expiration is almost entirely passive, so the weight of the arms on the chest tends to make it more effective and helps to counteract the extra inefficiency of expiration caused by relaxation under the anaesthetic. The net result is to improve and not to interfere with respiration. A small cushion should be placed under the lumbar spine to prevent it flattening under the anaesthetic.

I cannot claim to be an expert on this subject, but I have yet to hear any refutation of the simple mechanical and physiological principles on which my remarks are based or to see any willingness to carry out the simple changes in technique which they suggest.—I am, etc.,

Winsford, Cheshire. W. N. LEAK.

SIR,—In his recent article Dr. Cyriax (December 23, 1950, p. 1434) emphasizes the value of manipulation as a therapeutic procedure. Manipulable diseases are common in general practice, and in a practice of average size about 100 cases of such diseases are to be seen each year, if we include under this head most cases of fibrositis, acute torticollis, intercostal neuralgia, pseudo-angina, lumbago, and low backache, and also certain common types of neuritis, notably ilio-hypogastric neuritis, and certain painful conditions affecting the limb joints. Anyone who has to drive a car all day, every day, can sympathize with the indictment of the car-driver's seat; but the sagging bed can, I think, take priority as the commonest aetiological factor in the production of structural weakness in the lower part of the back. We spend one-third of a lifetime splinted into an awkward shape by bed-springs so designed as to ignore the fact that the hips are heavier than the feet and therefore sag much below the level of the feet in most beds—as any practitioner may observe on his rounds.

The contention that manipulation should normally be carried out without anaesthesia is undoubtedly a sound one, for, while there is a small group of cases in which manipulation under anaesthesia is the procedure of choice, there is no good reason for manipulating under an anaesthetic a joint which can be painlessly and safely manipulated without one. By using skill rather than force manipulation becomes one of the safest of procedures.

To discuss the theoretical aspects of manipulable diseases is to venture on dangerous ground. It is not at all self-evident that the lumbago and low backache so commonly met with in general practice should regularly be attributed to a prolapsed disk. Prolapsed disks undoubtedly exist and give rise to certain symptoms, but it is a far cry from this proposition to the claim that they are the usual cause of such symptoms. On the prolapsed disk theory it is difficult to explain the tenderness over the sacro-iliac joints in the common type of low backache one meets. Lumbago and low backache are usually bilateral, and, when treated by manipulation (see my letter, *Journal*, 1949, **2**, 710), one side is manipulated and then the other. After one side has been manipulated successfully, the pain remains on the other side of the body only. This observation appears to be quite incompatible with a disk aetiology for the condition, but it would be explained if a bilateral structure, such as the sacro-iliac joint, were to be incriminated as the source of the pain.

Joint manipulation has been, and still is, a somewhat empirical study; though the theory tentatively advanced by J. Mennell (*Joint Manipulation*, vol. 2) postulating the occurrence of an intra-articular "adhesion" (a seizing-up of articular surfaces opposed to one another) would seem to account for most of the phenomena met with in this field.—I am, etc.,

Lowestoft. N. B. EASTWOOD.

SIR,—In his interesting article (December 23, 1950, p. 1434) Dr. J. Cyriax offers useful advice on how to avoid the condition and how to prevent recurrence.

Surgeons are themselves sometimes affected. I have had one attack of sciatica considered by an orthopaedic surgeon to have been probably due to a disk protrusion, and many attacks of lumbago which I now believe were not produced by local chilling or other such causes, but were also of disk origin. Following some months off duty, I got sudden twinges of an attack of lumbago during the course of one of the first operations which I performed, an operation lasting more than an hour and which inevitably involved a considerable degree of spinal flexion. This unpleasant occurrence made me suspect that the strained position had caused the partial backward displacement of a nucleus pulposus. I was thoroughly alarmed, for I feared that a further period of incapacity was about to begin. I decided to try the effect of extension of the spine by weight. A folded towel was placed on the top of a door, and, hooking my fingers over this, I hung for a few minutes, and thereafter completed the operation without further trouble. This made me reflect how useful it would be if every surgeon's dressing-room had in it a three-foot length of horizontal bar. At the end of a morning's operating a short swing from this might well give a disk which had become partially compressed a chance to readjust itself. I wonder if Dr. Cyriax considers that such a simple piece of equipment might find favour with industrial medical officers. Since the date of my experience I have been careful when doing similar operations to pause from time to time and to stand in a very erect position, best described as making an attempt while one's heels are on the floor to touch the ceiling with one's head. And as I have now enjoyed an exceptionally long period without any attacks of lumbago I am inclined to believe that both diagnosis and treatment have been correct. None the less, I would welcome a piece of horizontal bar, and this would also save the door hinges.

—I am, etc.,

London, N.W.1. R. OGIER WARD.

Heart Disease and Mass Radiography

SIR,—Dr. Peter Leggatt's experience (December 16, 1950, p. 1364) of cardiological case finding by means of miniature mass radiography is of considerable interest to chest physicians and others who may have to make a preliminary diagnosis from an *x*-ray film without first examining the patient. He points out how an abnormal cardiac outline may be produced by rotation of the patient or by thoracic scoliosis, either cause being usually evident on the miniature film. A not infrequent cause of abnormal prominence of the pulmonary cone, which he does not mention and which should always be borne in mind, is depression of the sternum. Recently two patients have been referred to me by units with a report that, in addition to minimal tuberculosis, the *x*-ray film showed a "cardiac outline characteristic of mitral stenosis." Both patients had healthy hearts, but funnel-shaped depression of the sternum. In our diagnostic fluoroscopy clinic we have ample opportunity of confirming Dr. William Evans's teaching on the characteristic appearance of the heart shadow produced by sternal depression. One notices in particular the displacement of the heart to the left, and its rotation on the long axis, so that little if any cardiac shadow appears to the right of the vertebral column, and the left border of the heart is straighter than normal owing to prominence of the pulmonary cone. There is no increase in the transverse diameter of the cardiac shadow. The appearances, I believe, are sufficiently characteristic for the presence of a depressed sternum to be predicted with confidence, even though it is not usually visible on the antero-posterior film. My reason for drawing attention to these appearances, which are well known to cardiologists, is that the effect of this relatively common thoracic malformation on the shape and position of the heart shadow would not seem to be fully appreciated by all physicians in charge of mass radiography units.—I am, etc.,

Southend-on-Sea. E. G. SITA LUMSDEN.

SIR,—The many cardiovascular abnormalities encountered in the day-to-day work of the Glasgow mass radiography unit so impressed us that a survey of their significance was commenced in September, 1945, and continued till March, 1947. Our results were published in the *British Heart Journal*, 1949, **11**, 264. We were surprised, therefore, to note Dr. Peter O. Leggatt's comment (December 16, 1950, p. 1364) that "no one so far has attempted to assess the potentialities of this method for disclosing the presence of cardiac abnormalities." We recalled for clinical examination not only those who gave an abnormal radiological picture but in addition all those who gave a history suggestive of previous rheumatic infection, or of believing they had heart disease. It was hoped in this way, with the minimum of inconvenience, to ensure the detection of cardiac abnormalities in patients unaware of an existing heart lesion. Only the relationship between the radiograph and clinical findings is relevant to Dr. Leggatt's article. The most common causes of recall on account of *x*-ray abnormalities were general cardiac enlargement, prominence of the pulmonary artery, straightening of the left heart border, prominent conus, unfolding of the aorta, broadening of the base of the heart, and dextrocardia; of these the commonest were cardiac enlargement, prominence of the pulmonary artery shadow, and prominence of the conus. Of the 34,918 routine *x*-ray pictures of the chest in the period under review, 877 with no history of heart disease were examined because of one or other of these latter *x*-ray

abnormalities, and of these 44 were found to have organic valve disease and 8 congenital heart disease, a higher incidence than the 5 cases of organic valve disease and 1 of congenital heart disease found in the Liverpool survey. On the other hand the higher incidence in our investigation may be influenced by the fact that a history of previous rheumatic infection was also taken into account. Indeed, of the 44 cases of organic valve disease, 23 gave such a history. We have considered cardiological case-finding by mass miniature radiography to be sufficiently worth while to continue with this part of the service.—We are, etc.,

ALEX. MACLEAN.
A. S. ROGEN.

Glasgow.

Emotions and the Skin

SIR,—The criteria for the selection of patients with skin disorders for treatment by abreactive techniques (December 9, p. 1300) would serve equally well for the selection of patients for any psychiatric treatment. The skin conditions described by Dr. H. J. Shorvon and his colleagues were manifestations of psychosomatic illness and called for treatment by normal psychiatric methods. The fact that abreactive techniques were used is neither here nor there. Certainly there is, so far, no indication for the treatment of psychologically determined skin disorders by a specific form of psychiatric therapy.

Abreactive technique is a form of psychotherapy and not a manœuvre divorced from narco-analysis. Patients should not be abreacted merely in the hope of relieving them of tension. Tension is relieved by giving them an opportunity of expressing their emotions. They then become more capable of discussing their problems and viewing them objectively. Narco-analysis is a useful method of gaining a more rapid insight into personality and the possible factors underlying a neurosis. In 1941 I discussed (*Lancet*, 1941, 1, 507) the management of excessive abreaction in the course of narco-analysis, and I do not yet advocate uncontrolled discharge of emotional tension. Shorvon and his colleagues, on the other hand, emphasize the degree of excitement produced under drugs as the item of greatest therapeutic value, and they endeavour to excite their patients even beyond the natural and spontaneous release of inhibitions which occurs during narco-analysis. An opportunity to let off steam will relieve tension, and relief of tension may help a variety of skin conditions, but lasting benefit can be brought about only by the scientific application of abreaction. Deliberately to distort events in the patient's past history to produce greater excitement or anger appears to me to be very unjustifiable and possibly dangerous. Shock tactics may have to be adopted in some cases but surely should not be advocated as a routine practice.

I am surprised that some of the cases referred to in Dr. Shorvon's article were treated by prolonged and deep sedation before the abreactive method was attempted. Heavy sedation is invariably toxic in some degree, and as a treatment is usually contraindicated in psychological skin disorders. The case histories were most interesting, but demonstrated the value of narco-analysis more effectively than that of abreactive technique.—I am, etc.,

London, W.1. ELLIS STUNGO.

Containers for Syringe Sterilization

SIR,—The sterilization of syringes by dry heat as recommended by the M.R.C. Report of 1942 is still, in this country, the method of choice when conditions permit. In running a hospital syringe service the biggest problem is the means of packing syringes for sterilization, distribution, and return. It contains the answer to the question of breakages.

The method given in the M.R.C. Report is unsatisfactory. It consists in putting a syringe into a glass test-tube so that its flange rests on the lip of the tube and is retained by some form of wrapper. Once used the syringe cannot be retained in its tube. It is a common sight where this method is used to see dishes with a jumbled mass of syringes and empty tubes ready to be returned for servicing. The liability to breakages is increased by this procedure.

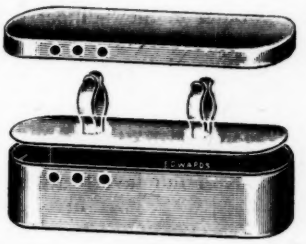
The writer has made an extensive trial of packing syringes in glass tubes made to size, so that each size of syringe is contained within its tube, retained by a cotton-wool plug and with a guard over its nozzle. Each syringe, after use, is replaced in its container and retained by its cotton-wool plug. It is thus protected until returned for servicing.

The disadvantages of this method are:

1. The excessive cost of tubes made to order.
2. Cotton-wool is generally a nuisance where syringes are concerned. Strands of the fibre can cause jamming of a well-fitting plunger, and can prevent the air-tight fit of a needle. Plugs, if used more than a few times, create a cloud of fine dust on disturbance, as shown by blood films made at the time. They are a small but continuous item of cost.
3. All containers made of glass are liable to break.

These objections are met by the use of the extruded aluminium containers illustrated here.

Each syringe is retained by malleable clips fixed to a thin-gauge sheet of aluminium which is a sliding-fit in the body of the box. A needle can be fitted before removing the syringe from the clips, so providing a stand until required. This is a great advantage on an anaesthetic trolley.



There is no cotton-wool. The lid of the box is sealed by adhesive tape, which can be quickly wrapped round the middle of the box after use, retaining the contents and indicating that they have been used. The boxes, not being cylindrical, cannot roll; they are much lighter than glass tubes of a comparable size; they are practically indestructible; and they are compact, being little longer than the size of syringe for which they are designed. The ability to sterilize a syringe with needle fitted is sacrificed, but this is, anyway, only satisfactory on metal nozzles, and leaves the operator without a choice of needle. Where dry-heat sterilization is not available these containers can be used in an autoclave. For this purpose holes are drilled, as in the illustration, and can be afterwards closed by turning the lid round.

While these boxes are very much cheaper than glass tubes made to order they cost a little more than those of standard sizes, but this is outweighed by their advantages and the fact that they do not break. They are made in four sizes, for 1 and 2 ml., 5 ml., 10 ml., and 20 ml. syringes. These boxes can be obtained from Messrs. Edwards Surgical Supplies Ltd., 83, Mortimer Street, London, W.1.—I am, etc.,

Hemel Hempstead, Herts. G. A. MATTHEWS.

Anaesthesia of the Newborn

SIR,—I was most interested in Dr. G. Jackson Rees's excellent article (December 23, 1950, p. 1419). By ignoring the relative lengths of trachea involved, however, I feel that his figure of $256x$ for the infant's resistance is excessive. Referring to Macintosh and Mushin's *Physics for the Anaesthetist* (p. 117), following the experiment which confirms Poiseuille's law, the authors halve the length of the tube involved and so double the flow. Taking the average glottis-to-bifurcation length in the adult as 4 in. (10 cm.) and in the infant as 2 in. (5 cm.), this would modify the resistance figure in the latter's trachea to $\frac{256x}{2}$, which although still high is a slightly less alarming figure than the original.—I am, etc.,

Dewsbury, Yorks. D. F. REES.

Bacteria in Ice-cream

SIR,—Your annotation (December 23, 1950, p. 1341) refers to the practice common among retailers of keeping serving-spoons immersed in jars of water which may easily become contaminated in warm weather, thereby undoing the good work which has gone into preparing a hygienic product. As this is a matter which concerns medical officers of health,

sanitary inspectors, and all those whose responsibility it is to advise people on hygienic practices, I have thought that it might be interesting to mention a simple but effective safeguard which we have adopted in our stores throughout the country as the result of a fairly intensive study of the problem over the past two years.

Wetting the serving-spoon or scoop is a necessary preliminary to serving in order to prevent the ice-cream from sticking, and, as running water is quite often not available at the serving point, a container or "dipper well" of tap water is kept at hand for this purpose. Our bacteriological records show that, provided the water is discarded every 20–30 minutes and replaced by fresh cold tap water, the danger of contamination is practically eliminated, and we recommend this practice to all concerned. No antiseptics or detergents are used. Containers and serving tools are sterilized daily.

We also apply a simple test which gives a rough but quite useful check on whether or not the water is being changed every 20 minutes. By measuring the total solids content of the water in the "dipper well" at appropriate times of the day, we get a pretty good indication of the state of hygiene discipline at the serving point. It is, of course, a truism to add that the best guarantee of good hygiene discipline is conscientious people who appreciate the importance of hygiene and who carry out the rules.

For those who are interested, we shall be glad to provide details of our investigations.—I am, etc.,

ALEC LERNER,
Chairman, Hygiene Committee,
Marks and Spencer Limited.

London, W.1.

Poliomyelitis in London in 1949

SIR,—The interesting article by G. E. Breen and B. Benjamin (December 30, 1950, p. 1473) unfortunately does not state in figures the cases in each borough on which the mathematical results have been worked. If, however, the map which is labelled "1949 poliomyelitis epidemic: corrected notifications" is taken as the basis for the figures I can only say that for Lewisham this basis appears to be wrong. I have counted the dots on the map and they come to 50 for this borough. In Lewisham, however, in 1949 there were 35 confirmed cases of poliomyelitis, and there were also 6 cases which ended up with a diagnosis of lymphocytic meningitis (presumed non-paralytic poliomyelitis). If the figure of 50 has been taken as the basis for Lewisham this shows, compared with the correct figure, a discrepancy of 20% or 40% according to whether the presumed cases are not or are respectively considered as confirmed cases. The above probably indicates one of the difficulties inherent in aggregating indirectly the figures of a number of epidemiological authorities.

There is just one other point in the article on which I should like to comment. That is the statement that the week ending June 18 is taken as the first week of "epidemic rise." In that week there were two cases in this borough, in sibs, and since their dates of onset were on consecutive days it is assumed they must have been infected from a third person earlier. Following them our next cases were reported on June 24, July 7, and two cases on July 11, these last from a local institution.

Perhaps it would be as well for me, here and now, to give the corrected figures for the last four years, including the one which has just ended. They are:

	Confirmed Cases	Presumed Cases
1947	76	7
1948	9	2
1949	35	6
1950	32	6

The last confirmed case notified in 1950 was on November 7, and therefore it is unlikely that any rediagnosis will be made.—I am, etc.,

E. H. R. SMITHARD,
Medical Officer of Health.

London, S.E.6.

Tuberculosis and the Health Service

SIR,—When Dr. J. Brailsford (December 30, 1950, p. 1493), a successful radiologist, eyes the report of the B.M.A. Tuberculosis Group Committee with suspicion, asking whether the dominant note is not self-interest, his comments should be scrutinized with the closest attention.

He italicizes sentences from your leading article of December 16 (p. 1373), but veils their significance. He appears to welcome the small reduction in the mortality from tuberculosis in recent years, but does not say that it is largely due to the short extension of life which chemotherapy has given to moderately advanced cases. In other words, chemotherapy delays death in many instances, and the graph of mortality is assuming a new shape. "That the average age at the time of infection with tubercle bacilli is rising" is not a sentence meriting excited italics but one to arouse the sober thought that parents and wage-earners are acquiring a chronic disease and becoming potential infectors of young and old. "That the expectancy of life of tuberculous persons is lengthening" is quoted as if it implied a major advance, whereas in sad truth it only means we have yet another problem to face in the case of the tuberculous. And Dr. Brailsford does not choose to underline, "that the number of new cases is increasing," presumably because it proclaims unanswerably the inadequacy of the present tuberculosis service.

On the other hand, the proposals of the B.M.A. Committee for the provision of properly equipped chest clinics, of hospital and sanatorium beds, and for increases in medical, nursing, and clerical personnel are based on knowledge gained by experience. A large part of the work required in the tuberculosis field is not being done, and cannot be done until the staff and equipment are provided. In one clinic in this capital of the world it takes up to three weeks to get an *x*-ray picture taken, and longer to achieve a bronchoscopy. There are insufficient health visitors to investigate the basic problem of contacts, and there are insufficient clerks to permit letters to go out to family doctors with proper dispatch. Shortage of beds in hospitals and sanatoria has forced on chest physicians the method of domiciliary treatment. This is only a second-best when compared with the advantages of investigation and treatment in hospital. It is an interim measure urgently devised to circumvent the shortage of beds, the feverish need for which it can never lessen.

But it is unfortunate that the B.M.A. Committee did not deal more critically with the sources from which working-class tuberculous patients can obtain financial assistance. Indeed, from the way they express themselves in your issue of December 16 (p. 1385) the impression is gained that plentiful aid can be obtained from a variety of sources. In actual fact a tuberculous patient who has to rely on the National Assistance Board and other agencies can support his family on only a very low standard of living, while renewal of clothing or household goods is rarely feasible. In other words, a man is, in effect, penalized for contracting tuberculosis. Many doctors speak disparagingly of what is called the Welfare State, implying it is altogether too bountiful. I have not yet sighted the coast of this mythical land. On the other hand, I am appalled daily by the parsimony of such aid as my patients can obtain.

I can agree if Dr. Brailsford believes that good housing and nourishing food are fundamental in the control of tuberculosis. But equally so are all the other factors mentioned in the B.M.A. report. May I, then, ask what steps are now proposed by the B.M.A. to achieve the realization of its recommendations?—I am, etc.,

L. N. GRUNBAUM.

London, N.W.8.

Electric Blanket Rash

SIR,—The patient was an elderly lady, aged 78, who was complaining of intense irritation on the extensor surfaces of the legs and arms. There was a papular rash over these areas. The papules appeared to be due to inflammation of the hair follicles. It was thought that she was suffering from some allergic reaction, possibly to a chemical soap used in the laundry.

The rash was resistant to all the usual treatments and the state of the patient was giving rise to some alarm, as she was getting no sleep. During one of the frequent examinations, a spark was seen to pass between the patient's shin and a loupe. On running the hand over the shin, several small shocks were felt. Only then was it discovered that the patient had acquired an electric blanket, which afforded her

much comfort. There was obviously a defect in the wiring of the blanket, with the result that the patient was shorting through a heavy blanket, which was directly in contact with the parts affected and which was also touching the iron framework of the bed.

The rash cleared completely when the electric blanket was banished.—I am, etc.,

Weymouth. ELLIS PARKINSON.

Long Umbilical Cord

SIR,—A few months ago I attended a patient in her third confinement. Everything was normal. The umbilical cord measured from its root in the placenta to the baby's navel 4 ft. 8 in. (142 cm.). There was one twist completely round the body, up over the front of the body, over the shoulder, under the same axilla, then up and twice round the neck, and a good bit to spare afterwards. Jellett's *Midwifery* says that the cord can be as short as five inches and as long as five feet, just in a general way. I think that this case must be more or less a record.—I am, etc.,

Valparaiso, Chile, South America. S. M. WELLS.

Helping the Newly Blind

SIR,—May we, as administrators respectively of St. Dunstan's and the National Institute for the Blind, draw the attention of those of your readers who may be asked for advice by patients losing their sight to the proper course newly blinded people should follow if they wish to take full advantage of the Blind Welfare Services?

If a person's loss of sight has been caused or aggravated by war service (including civil defence), he or she should get in touch with St. Dunstan's, 191, Marylebone Road, London, N.W.1, which is responsible for such cases.

Responsibility for all those whose loss of sight is not attributable to war service—that is, the civilian blind—is borne by the local authorities and the local voluntary agencies for the blind, in co-operation with the National Institute for the Blind. Newly blinded civilians should therefore get in touch with the local Blind Welfare Authority or, should there be any doubt about the identity of that body, with the National Institute for the Blind, 224, Great Portland Street, W.1, which gladly provides applicants with any information or advice they may require. —We are, etc.,

W. ASKEW,
Secretary, St. Dunstan's.
J. C. COLLIGAN,
Secretary-General,
National Institute for the Blind.

London, W.1.

POINTS FROM LETTERS

Unusual Presentation

Dr. A. J. MACLEOD (Lochmaddy, Isle of North Uist) writes: Delay in a recent labour in a primipara, which appeared straightforward, was caused by limitation of flexion of the foetal head. This proved to be due to the right forearm being across the throat, under the chin, bound down there by the cord, which was tight round forearm and neck. At birth the right hand was below and behind the left ear, the back of the hand next the neck. The descent of the head was slow, but delivery presented no difficulty.

Mercy Administrator?

Dr. B. D. JOHNSON (Leicester) writes: We seem to live in an age when it is becoming increasingly unfashionable to call a spade a spade. I find in Dr. C. K. Millard's letter (December 23, p. 1447) on euthanasia such terms as "euthanisors" and "mercy administrators" advanced as suitable for those who, it is proposed, are to be licensed to kill patients suffering from "incurable illness." I would have a far greater respect for Dr. Millard and his society (with whose views I profoundly disagree) if he expressed his meaning in simple terms such as "patient killers" . . . rather than hide his meaning in unhappy euphemisms or ill-conceived neologisms.

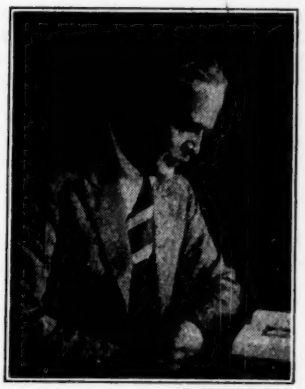
Correction.—In Mr. McNeill Love's letter on appendicitis (January 6, p. 36) the first line of the penultimate paragraph should read, "As admitted in the article, appendicectomy is *not* always possible. . . ."

Obituary

G. R. GIRDLESTONE, D.M., F.R.C.S.

Mr. G. R. Girdlestone, who played a leading part in the advancement of orthopaedic surgery in this country, died on December 30, aged 69 years.

Gathorne Robert Girdlestone was born in Oxford in 1881; his father was Canon R. B. Girdlestone, of Christ Church, Oxford. From Charterhouse he went on to New College, Oxford, and at St. Thomas's Hospital, where he continued his medical education, he was University Scholar. He qualified M.B., B.Chir. in 1908, and, after serving as casualty officer and house-surgeon at St. Thomas's, he went to Oswestry as a partner in a surgical practice, and there he immediately came under the influence of Sir Robert Jones, who regularly visited the hospital for crippled children which Dame Agnes Hunt had established at Baschurch. It was as Sir Robert's lieutenant that he played such an important part in the founding of the Central Council for the Care of Cripples. In 1911 Girdlestone became a Fellow of the Royal College of Surgeons, and not long after the outbreak of the first world war he was appointed, while a captain in the R.A.M.C., to take charge of a unit for the treatment of bone and joint injuries at Oxford. Those few huts at Headington were the origin of the world-famous Wingfield-Morris Orthopaedic Hospital. It was Girdlestone's enthusiasm and skill as a surgeon which were largely responsible for Lord Nuffield's immensely practical interest in promoting this hospital. No less valuable than the hospital itself was the system of clinics which linked it to towns and villages in Oxfordshire, Buckinghamshire, and Berkshire. Girdlestone travelled widely to explain the advantages of this method of organizing orthopaedic care, and it was after a visit to South Africa that Lord Nuffield established an orthopaedic trust there similar to the trust he formed for the development of orthopaedic work in Great Britain and Northern Ireland.



[*Lafayette, Ltd.*

Though the Wingfield-Morris Hospital was the main interest in Girdlestone's life, he built up a large orthopaedic practice and was on the staff of many hospitals, including the Radcliffe Infirmary, the King Edward VII Hospital at Windsor, the Royal Buckinghamshire Hospital at Aylesbury, the Savernake Hospital at Marlborough, and the Robert Jones and Agnes Hunt Hospital at Oswestry. The American Orthopaedic Association elected him a corresponding member. In 1937 he was appointed to the first chair of orthopaedics to be established in this country, the Nuffield professorship of orthopaedics at Oxford. He did not remain professor long, however, for he resigned in 1940 and became a consultant to the E.M.S. and to the Ministry of Pensions. In 1942 he was elected President of the British Orthopaedic Association. He wrote many articles in journals and textbooks, and his book, *Tuberculosis of Bone and Joint*, was published in 1940.

We are indebted to Professor A. D. Gardner for the following appreciation: Wherever in the world orthopaedic surgery is practised the name of Girdlestone is known. He was prominent in the small group of pioneers who developed, improved, and popularized in Great Britain and the Dominions the principles and methods of the prime originators of this practical science, which has transformed the lives of innumerable deformed and injured human beings. His special teacher and example was Sir Robert Jones, whose methods he studied in collaboration when he was the surgical partner in a firm of doctors in Oswestry. Much also he owed to Dame Agnes Hunt, whose hospital at Baschurch provided him with an early model of what he was eventually to create himself in Oxford. It was by no means only the important advances that he learned there in the treatment of crippled limbs, but also the spirit of devotion and selfless love of humanity with which in later years he inspired all his associates. He himself was a born surgeon, with strong, sensitive hands and an imaginative insight into the distresses of the living structures he was handling. Never-ceasing study and perpetual contact with experts in all branches of medicine made and kept him a master of his subject, to which he made many notable contributions. These included descriptions of original operative methods, systematic clinical observations, and an important monograph on tuberculosis of the knee-joint. On the organization of orthopaedic work in hospital and clinic, and on the spirit in which it should be done, he spoke and wrote continually; and it is perhaps in this aspect of orthopaedic surgery that his most massive and original contribution lay. The organized system of satellite clinics, surrounding and served from the central hospital, which he created in the Oxford region will long remain the model not only for orthopaedics but for all forms of specialist regional services. Looking back over the contracting years one seems to see "the Wingfield" growing like magic around him, and spreading its healing influence further and further into the counties. It was not long before Lord Nuffield fell under his spell and gave the splendid aid which transformed the temporary hutments of the nineteen-twenties into the famous Wingfield-Morris Hospital of to-day. A further recent benefaction has secured Girdlestone House, for many years his home on the outskirts of the hospital, as a valuable residential annexe and a permanent memorial to his name.

"G.R.G.'s" many-sided character gained him innumerable friends. He was a superb golfer, who might well have been amateur champion if he had not had better things to do, and he excelled at all games requiring a keen eye and accurate control. On his professional side he became in due course President of the Orthopaedic Association. When Lord Nuffield's great medical benefaction came to Oxford in the nineteen-thirties he was the only possible choice for the chair of orthopaedic surgery, which he occupied with much distinction. But after some years of increasing strain he found it necessary to return to the life of clinical practice and hospital organization which he loved and of which he had made such a brilliant success. His loss as a person is irreparable, but his 69 years of life have been very full of achievement and will long remain an example and enduring influence.

H. J. S. writes: Girdlestone's life was the Wingfield-Morris Hospital. His devotion to it dwarfed all his other achievements and his many talents. This generous and affectionate man was childless, and so he cherished with parental care the miserable collection of huts in which he started his career as an orthopaedic surgeon until, in the fullness of time and with the inspired aid of Sir William Morris, as he then was, it blossomed out as one of the premier orthopaedic hospitals of Great Britain. His solicitude extended to every member of the staff; in sickness, in family troubles, and in financial difficulties he was ready to help, and no one can catalogue his many acts of kindness. There was a remarkable atmosphere about the place that made for happiness and efficiency, and the best witnesses are the host of old patients who are deeply attached to the hospital and to its staff. At the beginning of the second world war it was proposed that a large number of E.M.S. huts should be added to the Wingfield, and the plans were actually drawn. The moment Girdlestone learned how much some hospitals had suffered as a result of enforced expansion he never rested until he had found a way of getting the additional beds without detriment to his own hospital. He explored every plot of open land in Headington; he found a good site; he prevailed on the Ministry of Health to build a decent-looking emergency hospital in brick; and he worked with the architect on every detail of the plans. The result was that Oxford was given a new hospital—the Churchill—which has proved invaluable since as well as during the war. Then came the question, who was to work there? That excellent corps of volunteers called the American Hospital in Britain was first housed a little uncomfortably at Park Prewett, near Basingstoke. Why not give them a whole new hospital to themselves? So they came to Oxford. The Stars and Stripes and the Union Jack were run up one day in a driving snow-storm, and, thanks to Girdlestone, that very gallant company of Americans, who came here during the darkest period of the war, was able to work in congenial surroundings. This was but one instance of Girdlestone's creative ability and tenacity of purpose. Whatever he did was be important a clash was inevitable, for there was no done with all his might. When he encountered colleagues who differed from him on some matter that seemed to possibility of compromise; but even in the heat of battle it was impossible not to admire so staunch a fighter. And it was a great privilege to have worked with him.

Professor J. Trueta writes: The hazards of my life have brought me in contact with many men, but I cannot recall any who, apart from my father, had more influence on my life than "G. R. G." When we first met shortly after my arrival in England almost twelve years ago he extended to me his protective arms, brought me to Oxford, and watched attentively all my steps until he saw me taking roots in this country and in the hospital he had founded. This intimate friendship and daily contact gave me the opportunity of discovering one of the most extraordinary personalities of the medical world of to-day. He was a man of whom any country, at any time, might be proud. As in all men of real genius his great qualities seemed to be his only salient defects. By his simplicity he failed to impress superficial observers, and by his modesty and retiring disposition he never attained the recognition to which his great deeds entitled him. His modesty was apparent to the end by his refusing to have a memorial service dedicated to him. His directness, unfailing purpose, and sincerity, together with his incapacity for dissimulation, were a source of suffering to him and sometimes to others. But all these attributes were like small gems implanted in a

golden heart. Here lay his strength and the outstanding feature of his personality.

Girdlestone has been an example of a man who lived to love. He loved God and all that comes from Him. He loved his country, his friends, his patients, his profession, and his hospital. As a summation of all his other great affections he concentrated his tenderness on his wife, Ina, the faithful companion of his life. The stream of crippled and infirm children who came under his care for more than forty years were as their children, and he nursed them with the tenderness of a father. Love brings forth love and also fruit. In 1919 the pages of this *Journal* were honoured with the publication of an article on "The Cure of Crippled Children," signed by Sir Robert Jones and G. R. Girdlestone. There an almost Utopian plan for the prevention, treatment, and education of children suffering from orthopaedic conditions was expounded by the master and the young reformer. Now, thirty years later, the Wingfield-Morris Orthopaedic Hospital and the orthopaedic organization of the Oxford Region stand as a lasting monument to the memory of the genius who made Utopia a reality. He imparted to this hospital so much of his love that he leaves behind in it what is known as a "Wingfield spirit." The only recompense for which he sought was the affection of his patients and of those who surrounded him, and in that he was amply rewarded. We, his intimate friends, have suffered an irreparable loss, his patients lose a great comforter, and England a great social reformer.

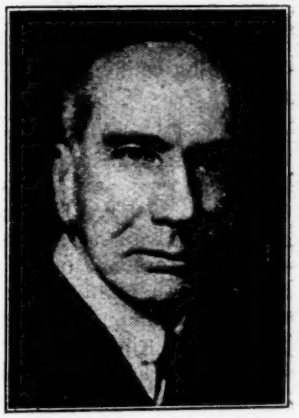
Sir LENTHAL CHEATLE, K.C.B., C.V.O., F.R.C.S.

One of the few remaining personal links with Lister is broken by the death, on January 2, of Sir Lenthal Cheatle, who had been identified with King's College Hospital—Lister's London hospital—for well over half a century. When he was surgical registrar at King's from 1889 onwards he came into close association with Lister, who occupied the chair of surgery at the College until 1892, and in fact Cheatle assisted him in the last operation that he performed.

George Lenthal Cheatle was born in 1865. He received his education at Merchant Taylors' School and at King's College and Hospital, qualifying in medicine in 1887. He was already a member of the Medical Society of King's College in the session 1885–6, and by 1889 he had become vice-president. He held a number of posts successively in his own hospital, which was then in the neighbourhood of the Strand—demonstrator of anatomy, house-surgeon, house-physician, surgical registrar, and, from 1892 to 1894, demonstrator of surgical pathology. His appointment as assistant surgeon at King's dated from 1893, and in 1900, the year he obtained the F.R.C.S., he was made full surgeon and teacher of surgical pathology.

In the early days of the Boer War he went out to South Africa with his former chief, Sir Watson Cheyne (the house-surgeon whom Lister had brought from Scotland), both of them receiving a great send-off from King's students at Waterloo. Cheatle was appointed consulting surgeon to H.M. Forces: he was mentioned in dispatches, received the campaign medal and four clasps, and was made Commander of the Bath. In the European war of 1914–18 he gave his services to the Royal Navy, holding the rank of surgeon rear-admiral and serving at Haslar and in a hospital ship during the Gallipoli campaign. At the end of the war he was made K.C.B. He continued for many years to be a member of the Consultative Board of the Royal Naval Medical Service and of the Admiralty Departmental Committee. Sir Lenthal Cheatle also took an active part in the creation of a medical branch of the Naval League.

On his return from naval duties his work for King's College Hospital was resumed, and in 1923 he became senior surgeon and one of the lecturers on surgery. He retired from the active staff in 1930, and was appointed consulting surgeon to the hospital and emeritus lecturer on clinical surgery in the medical school. Among other appointments which he had held were those of surgeon to the Florence Nightingale Hospital, and consulting surgeon to the Hospital for Paralysis and Epilepsy, the Surrey Dispensary, and the Sevenoaks and Burford cottage hospitals. He had also held the office of president of the Section of Surgery of the Royal Society of Medicine. Honours were bestowed on him by the Italian and French Governments; by the former he was made an Officer of the Grand Cross of Italy, and by the latter Chevalier of the Legion of Honour. The American College of Surgeons made him an Honorary Fellow in 1932. In the previous year he had received the Walker Prize from the Royal College of Surgeons of England for his work on the pathology and therapeutics of cancer.



To the end of his life Lister was his great hero, though his personal association with him had been limited to the last few years of Lister's reign at King's. He spoke often of Lister's devotion to work and of his courtesy to all who assisted him, and was fond of telling how to Lister the operating theatre was a temple, of whose sanctity he would permit no infringement. One occasion he recalled was when Lister brilliantly performed an operation in 32 seconds, and when the onlookers broke into a subdued cheer he turned round and said, "Gentlemen, remember where you are." One outstanding characteristic of Lister which Cheatle strove successfully to emulate was scrupulous accuracy and attention to detail. He once told Cheatle that he could not recall ever having made an inaccurate statement, that there was nothing he had ever said or written which he wished to retract.

It was his association with Lister and Watson Cheyne that gave Cheatle his special interest in pathology. He was one of the most careful of investigators. Early in his career he recognized the importance of cutting and examining sections of an affected organ, and for this particular purpose he devised the large microtome sometimes associated with his name. This method of investigation he applied in particular to carcinoma of the breast, on which subject, in collaboration with Dr. M. Cutler, he wrote a textbook, *Tumours of the Breast*. He also wrote the section on inflammation and diseases of the breast in Sir James Walton's *Surgical Diagnosis* and the article on suppuration in Choyce's *System of Surgery*. He was one of the earliest to give injections of tetanus antitoxin to patients with wounds

contaminated by dirt. His earliest note on that subject was published in 1910.

For many years Sir Lenthal Cheatle was president of the King's College Hospital Lawn Tennis Club, to which he presented a cup for the singles championship open to students of the school. In the eighties he was for several years one of the most successful cricketers that King's College—never backward in sport—could put in the field.

He married Miss Clara Jopp in 1902, and had three sons and one daughter. Lady Cheatle died in 1942. His brother, the late Arthur Cheatle, F.R.C.S., one of the leading otologists of his time, was his colleague at King's.

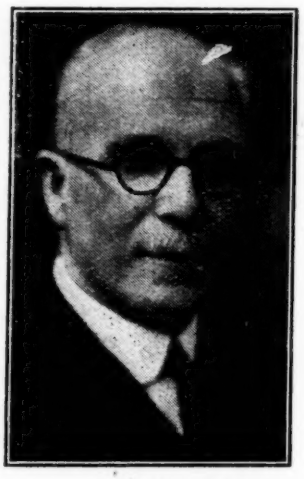
C. P. G. W. writes: It was while Cheatle was surgical registrar that he came in contact with Lord Lister, and he never missed a ward visit that Lister undertook. He was well versed in the Listerian method of dressing wounds, and he always remained a staunch antiseptic surgeon, using 1 in 40 carbolic for all his instruments. Lenthal Cheatle had a great personality: he was charming, suave, and good-looking, and he was a great favourite with the students, nurses, and patients. He always came to hospital wearing a morning coat and a top hat. Twenty years of retirement has meant that few of the younger generation of surgeons have heard of his work.

THOMAS FRASER, C.B.E., D.S.O., LL.D., M.B.

Dr. Thomas Fraser, one of Aberdeen's most notable medical men, died at his home on January 3 at the age of 78 years. Blessed by good health for the greater part of his life, he was able to carry on his large practice until just about a year before his death.

Thomas Fraser's father was the late Provost T. Fraser, of the ancient Royal Burgh of Kintore, within a short distance of Aberdeen. He was educated at Robert Gordon's College, Aberdeen, whence he proceeded to the University. As was the custom at that time, he entered the Faculty of Arts and graduated M.A. in 1894. Four years later he completed the medical course and graduated M.B., Ch.B. He then spent a short time in general practice in Leeds, returning to Aberdeen in 1900, in which year he obtained the diploma of public health with credit. About this time he established himself in general practice. One of his earliest appointments was in the old Aberdeen Dispensary, at which attendance by medical students was compulsory: they not only had to attend out-patient clinics but also visited patients in their own homes. Some time thereafter began his long association with the Aberdeen Royal Infirmary by his appointment as assistant anaesthetist, then the recognized stepping-stone to the honorary staff. Later he became assistant physician, and in 1919 full physician and lecturer in clinical medicine to the University. Finally, on his retirement from the hospital in 1932 he was appointed consulting physician. At one time he also held the post of obstetric physician at the Maternity Hospital. At an early period in his career he became attached to the materia medica department of the University, first as assistant and later as lecturer to the late Professor Cash. He became interested in the problems of diabetes, and collaborated with the late Dr. John Rennie, of the natural history department, carrying out some important researches on the aetiology of this disorder. Rennie had pointed out that in certain of the teleostean fishes the islands of Langerhans formed an almost separate organ. Extracts of these were made and fed to diabetic patients, and to one at least the extract was given by injection. This may quite truly be said to have been the precursor of insulin, but neither biochemistry nor the ancillary sciences were sufficiently advanced to allow of the development of this brilliant hypothesis.

As his practice was increasing he was compelled to give up some of his university studies. Always a keen Territorial, he served in the first world war as a lieutenant-colonel, commanding the 89th (1st Highland) Field Ambulance, and he was present at the landing at Hellas. Invalided from Gallipoli in 1916, he was later transferred to France as officer commanding 51 (Highland) Casualty Clearing Station. In 1918 he was appointed A.D.M.S. 47th (London) Division, with the rank of colonel. The war years had brought to light his great capacities as an administrator, and the brilliance of his wartime service is attested by five mentions in dispatches and by the award of the D.S.O. in 1918 and the C.B.E. in 1919. In addition he held the Territorial Decoration.



[MacMahon, Aberdeen

In the immediate post-war years the demands of his large practice and his hospital teaching left him with little leisure, and such spare time as he did allow himself was given over to the work of the British Medical Association and of local medical affairs. He was honorary secretary of the Aberdeen Branch from 1905 to 1927. In 1914 as joint secretary he was largely responsible for the success of the Annual General Meeting in Aberdeen. Besides serving on a number of the major committees, he was a member of Council from 1927 to 1939. He was elected President of the Association in 1939, and he retained this office until 1942. In addition, from 1941 to 1946 he was a member of the General Medical Council. The City of Aberdeen conferred upon him the distinction of Deputy Lieutenant. Of none of the many honours which came to him was he more proud than the LL.D. degree which the University conferred upon him in 1939—a fitting tribute to one who for so many arduous years had been a devoted and loyal son of his alma mater.

Perhaps he was the last survivor of a generation in which it was possible for a man to conduct the arduous duties of a general practitioner and at the same time be an outstanding teacher in a medical school, and to-day many of his former students will look back with gratitude on all they learned from him and will remember his courtesy, his dignity, his humanity, and the example of one who conducted all his work on the highest ethical standards. He is survived by his widow and two sons, to whom much sympathy is extended.—A. G. A.

W. C. S. writes: The passing of Dr. Thomas Fraser is not only the passing of a well-beloved family doctor but the passing of an era in general practice so far as the North of Scotland is concerned. Dr. Fraser's claims to medical fame were laid on very wide and solid

foundations. He was a fine example to the medical youth at the Aberdeen Dispensary of the combination of the bedside manner *par excellence* and the shrewd clinical observation that stamped him through all his wide practice. Always thinking of the patient—early or late, far or near, poor or rich—if the needs of the case were there so was he. Never did any physician spend himself more unselfishly for his patients. Whenever an operation was performed on any of them he made it his business to be there, so that he would know exactly what was present and what was done in each case. In this way he kept a very close watch and ward over all his cases and was thus able to assess more fully future developments from past experiences. For many years he enjoyed a fine reputation as an accoucheur, and his help was prized by many colleagues as well as by the patients. It was difficult to see how he could stand up to so much night work when his daily visiting lists were so long, particularly when one remembers how generous he was at any call—no hurried in-and-out for him. In all his professional dealings he was most correct and proper—indeed, even fastidious by modern standards—but he clung to etiquette and enjoyed great kudos for that. In the earlier days his voice was heard at medical festive occasions in song and, on more serious occasions, in speeches that were always well prepared and carefully delivered in the Asquithian manner. He was a voracious reader of all sorts of literature, and kept himself informed on all matters of current medical and general interest, while spending all day and often part of the night on his daily task, his patients. He was a student of the art and craft of prescribing and therapeutics, and elegance added to his courtesy at the bedside made him indeed the beloved physician; no trouble was too much—in fact, he wiped the word trouble out of his dictionary. Quiet dignity and efficiency inspiring confidence, delightful easy friendliness, and courtesy abounding combined to make him "a perfect gentle knight," and his memory will be long cherished by the laity and by the profession he did so much to adorn.

HERBERT STANLEY FRENCH, C.V.O., C.B.E. D.M., F.R.C.P.

Dr. Herbert French, formerly physician to His Majesty's Household, died in London on January 1 at the age of 75. He was at the time of his death consulting physician to Guy's Hospital, to Queen Alexandra's Military Hospital, and to the Royal Hospital at Chelsea.

Herbert Stanley French was born in Newcastle-upon-Tyne, where his father was a merchant. He was educated at Dulwich College and Christ Church, Oxford, to which he gained an open scholarship. It was at Christ Church that he met the son of Sir James Sawyer, a physician of Birmingham, whose daughter, Amy, he married in 1904. After obtaining a first-class in the National Science School he came to Guy's Hospital with a university scholarship in 1898, and there won the Beaney Scholarship, the Beaney prize for pathology, and the Gillson Scholarship of the Society of Apothecaries. He graduated B.M., B.Ch. in 1900, and, after holding the post of house-physician at Guy's, he held a Radcliffe Travelling Fellowship from 1902 to 1904, the year he proceeded D.M. In 1906 he was appointed assistant physician to Guy's Hospital and in 1917 full physician. He was admitted F.R.C.P. in 1907 and was Goulstonian Lecturer in 1908, choosing as his subject "The Medical Diseases of Pregnant Women." For many years he was physician to H.M. Household. During the first world war he served as honorary lieutenant-colonel in the R.A.M.C. and was appointed consultant to a large military hospital at Aldershot. He also became consultant physician to the Queen Alexandra Military Hospital, Millbank, the Royal Hospital, Chelsea, the Radium Institute, the Enfield Hospital, and the Surrey Dispensary. He was made a C.B.E. in 1919 and a C.V.O. in 1930. In 1928 he was president of the Harveian Society. Early in his career he examined in medicine for the Society of Apothecaries, and later for the University of Oxford and the Royal College of Physicians. He was the author of many articles in journals and textbooks, but in this field he achieved most fame as editor of *An Index of Differential Diagnosis of Main Symptoms*, which first appeared in 1912. He retired from professional life in 1929, when still in active middle-age, and devoted himself to his property in Surrey, where he turned with whole-hearted enthusiasm to fruit farming and cider and chutney making. His first wife died in 1946, and in 1948 he married Nora, widow of Edward McDonald. He leaves a widow, son, and daughter.

A. H. D. writes: French appeared never to have any doubts about his decisions or the goal for which he was making. His determination to succeed was evident in his student days, and as he passed through the successive phases of house-physician and registrar to assistant physician his quite extraordinary power of intense industry became more and more apparent. As a house-physician he started collecting notes, sketches, and photographs of cases of interest, thus providing the core of the book which was later to make him famous. Soon after election to the staff at Guy's he developed pulmonary tuberculosis and went for some months to Cornwall, where he devoted his time to sailing and fishing. Within a few months he was back at work, clearly recovering in spite of the most exiguous treatment as judged by modern standards. He delighted in later years to demonstrate his health and strength "in spite," as he said, "of 16 positive sputa."

He was a most impressive teacher, partly because of his love of the dramatic and of simile, partly because of the emphasis he would lay on the practical side of therapeutics. He was just as ready to teach the best way to insert a bed-pan as he was to discuss the latest development of the focal-sepsis theory. His post-mortem demonstrations and reports were a model of lucidity and an example to those who followed him. His clinical lectures always secured him a packed theatre, where he would without apparent effort speak and illustrate on the blackboard for an hour without notes and without hesitation. He prided himself on a teutonic meticulousness which made it possible for him to edit *An Index of Differential Diagnosis of Main Symptoms*. The index alone took a year to complete and entailed the making of over half a million entries. Most of this work was done when travelling in his car to consultations, for which he was in ever-growing demand. His other most important contribution to medicine was a description of influenza as it appeared in the 1918 pandemic. During these years he continued to extend the scope of his clinical experience while fostering an ever-increasing consulting practice, which by the end of the war was probably one of the largest in the kingdom. His health was now robust, and he was able to enjoy a full social life in addition to his professional activities. By 1920 the strain on his

capacity for work had reached fabulous proportions, and he had come to the stage of success when not only doctors but also laymen would ask for his help. He was a strict observer of etiquette in relation to professional work and never allowed the patient to consult him without the knowledge of the general practitioner.

With a characteristically dramatic gesture he retired at the age of 55, sold his house and everything in it, even to his stethoscope, and retreated to Newdigate in Surrey. Here he was able to enjoy the delights of country life and holidays on his yacht. He had taken his master's certificate, and grasped with his usual clarity of mind the intricacies of navigation. His health and strength seemed imperishable. The death of his wife a few years ago was a great blow to him, but he faced this with the same indomitable courage which he showed in his final illness. His happiness was restored when he married again some two years ago, but a painful and fatal illness struck him last autumn, and he ended his days in the hospital he loved and served so well.

GEORGE C. CATHCART, M.D.

Dr. George Clark Cathcart, who died in a nursing-home at Broadstairs on January 4 in his 91st year, practised as an ear, nose, and throat specialist for many years in Upper Wimpole Street and in his day had one of the best consulting practices in the West End of London.

Cathcart belonged to an old Edinburgh family, his elder brother Charles being a well-known teacher of surgery at Edinburgh, and he went to school at Edinburgh Academy and Loretto. He graduated M.A. at Edinburgh in 1883, and thereafter went to Australia and back in a sailing ship and was a pupil at Naples of Scarfati, one of the last teachers of the old Italian *bel canto*, in order to cure a stammer. He returned to Edinburgh to study medicine, graduating M.B., C.M. in 1891 and proceeding to M.D. in 1908. He came to London as a resident at the Children's Hospital, Great Ormond Street, as did many Edinburgh graduates of those days, and then became resident house-surgeon at the old London Throat Hospital (later amalgamated with the Throat Hospital, Golden Square), to which he was later appointed honorary surgeon. After the amalgamation he became consulting surgeon to the Throat Hospital, Golden Square, and he was also consulting laryngologist to Mount Vernon Hospital, in the days when it was still a hospital for tuberculosis. During and after the first world war he was a member of the Special Aural Board of the Ministry of Pensions.

In his earlier days Dr. Cathcart was greatly interested in problems of the voice, and at that time his contributions to medical literature were mainly on such subjects as voice production, stammering, and tonsillectomy. It was directly because of this interest that in 1894 he founded the famous Promenade Concerts at the Queen's Hall, London. He insisted that in order to prevent vocal strain the orchestral instruments should be tuned to French pitch and not to the higher concert pitch, as was then usual; and he also insisted that his young friend and "discovery," Henry Wood, should be the conductor of the series of concerts. Then a young bachelor of independent means, Cathcart was quite content, in order to prove his thesis, to lose several thousand pounds in starting the concerts, and when they began to pay their way he gave place to others. In his later days he became more and more interested in deafness and its prevention. He revised and largely re-wrote his friend Hunter Tod's *Diseases of the Ear* in 1926, and in 1931 published a small book, *The Treatment of Chronic Deafness*, which went into a second edition. Not infrequently the victim of his own kind heart, he was also ever sympathetic towards the unorthodox in medicine—homoeopathy, osteopathy, the electrophonoide method of Zünd-Burguet. He had always a current favourite among such methods and their practitioners. He was an ardent freemason, having been a Past Master of the University of Edinburgh Lodge and a member of Grand Lodge.

Dr. Cathcart never married, but he had many nieces and other relatives to whom he was devoted, and there can have been few men more beloved by more friends in every walk of life.—R. S. S.

Dr. ALMA PERCY FORD, who from 1919 devoted his life to the care of patients suffering from tuberculosis in the county of Hertford, died suddenly on December 18, 1950, at the age of 60. Born at Spalding, Lincolnshire, he was the son of Alderman A. Ford, J.P. After leaving the Mercers' School, he studied medicine at the London Hospital, where he qualified in 1914. On the outbreak of the first world war he volunteered for service in the R.A.M.C. and was sent to France in August, 1914, where he served until he was demobilized early in 1919. He took the D.P.H. in 1920, and after a short period as assistant school medical officer in Huddersfield he was appointed tuberculosis officer in Hertfordshire and also deputy county medical officer. He became the driving force behind the great development of the tuberculosis service in the county, and his work was recognized in his appointment as one of the two representatives of the British Medical Association on the Joint Tuberculosis Council. At the time of his death he was honorary treasurer of the Council, a position which he had held for many years. At the inception of the National Health Service Ford was chief clinical tuberculosis officer in Hertfordshire and medical superintendent of Ware Park Sanatorium. After July, 1948, he became a consultant chest physician and physician-superintendent of Ware Park Sanatorium. Always a keen member of the British Medical Association, he was honorary secretary of the East Herts Division from 1931 until 1938, and was chairman of the Division from 1939 until 1944, guiding the members through the difficult war years with outstanding clarity of judgment and tact. None of his friends and colleagues in the Joint Tuberculosis Council and British Medical Association will forget his forthright personality radiating from his powerful, well-built figure. His interests were wide—at one time he was captain of the London Hospital association football team; he held a lifelong passion for cricket; he was a keen golfer, holding the Ledward Cup for several years; and he was an active supporter of the Amateur Dramatic Society of Hertford. Ford contributed a number of papers to medical journals on various aspects of the treatment of tuberculosis and was joint author, with the late Dr. Hyslop Thomson, of *Tuberculosis of the Lungs*, published in 1927. His loss will be widely felt not only by his medical and nursing colleagues and his many friends, but by innumerable patients all over the country who have known his sympathy and dynamic energy. In 1915 he married Lilla Robeson, who with their two daughters survives him.—C. P. C.

The Services

The *London Gazette* has announced the following awards:

Bar to Air Force Cross.—Wing Commander H. P. R. Smith, A.F.C., R.A.F.

Air Force Cross.—Squadron Leader J. S. Howitt, R.A.F.

Universities and Colleges

UNIVERSITY OF OXFORD

In Convocation on January 16 it will be proposed to confer the honorary degree of D.C.L. on the Rt. Hon. Sir Godfrey Martin Huggins, P.C., C.H., K.C.M.G., F.R.C.S., Prime Minister of Southern Rhodesia.

UNIVERSITY OF SHEFFIELD

The Council of the University has decided that the new Hall of Residence for Men which is being erected in Oakholme Road shall be named Stephenson Hall in honour of members of that family who have served the University and its forerunners in successive generations.

Dr. D. E. Price has been appointed Honorary Lecturer in Pathology.

EPIDEMIOLOGICAL NOTES

Smallpox at Brighton

Up to noon on January 9 there were 17 patients believed to be suffering from smallpox in Brighton county borough, or in the immediate vicinity of these cases. Two have died, and the diagnosis in three was still in doubt. During the previous week the infection had been confined to the two original foci—the laundry, to which the clothing of the initial case, an R.A.F. officer, was sent, and the hospital, in which the second case was not diagnosed for 10 days.

Among the laundry employees there have been six cases. Two of these worked at a receiving branch and the remainder in the main establishment. The laundry has now stopped work. The hospital is in quarantine, and members of its staff admitted to the smallpox hospital consist of five nurses and one domestic. All these persons were employed on the ward to which the second case was admitted and nursed in a cubicle.

The vaccinal state of the nursing and domestic staff at the infectious disease hospital was unsatisfactory, 27 out of 113 never having been vaccinated. On investigation among nurses and domestics attached to the ward concerned, it was found that seven were unvaccinated. Of these, three had very little contact with the patient and did not get the disease. All the remainder had intimate contact, and one died and three are in a serious condition. The other two nurses now in the smallpox hospital have a good vaccinal history, and in their cases there is still some doubt about the diagnosis.

Poliomyelitis

Poliomyelitis notifications in the concluding week of 1950 were: paralytic, 36 (38); non-paralytic, 17 (15); total, 53 (53). The figures for the previous week are in parentheses. The total uncorrected notifications for 1950 are 8,696, compared with 9,195 in 1947, 2,379 in 1948, and 6,857 in 1949.

There were no notifications in the week under review from 35 county areas; and in the remainder notifications were more or less evenly distributed, no county notifying more than four.

The incidence of poliomyelitis in 1950, after correction of notifications, is hardly likely to be much less than that in 1947, when it was 4½ times greater than in any previous year since compulsory notifications began in 1912.

Influenza

The statistics given below for England and Wales and the Great Towns do not indicate any serious epidemic of influenza up to the end of the year. Any increases in pneumonia cases and deaths and influenza deaths are not much more than normal for the time of year. A mild form of influenza, but of a very infectious type, has been epidemic in the north-east of the country, including Newcastle-upon-Tyne and elsewhere, in parts of Lancashire, including Liverpool, and in Birkenhead (Cheshire). But in these districts the epidemic has probably passed its peak. Serological investigations have shown that the epidemic in Tyneside is due to virus A; it may be identical with that in the Scandinavian outbreaks—virus A prime. The results of further laboratory investigations are awaited.

There is at present no evidence of spread of the epidemic to the Midlands and South. But in many areas there are local outbreaks of a mild disease with symptoms not unlike those of influenza; they have not yet been shown to be due to an influenza virus.

There is serological evidence that in Sardinia cases of influenza have been due to virus B.

The following cases of and deaths from pneumonia and influenza have been reported in the Great Towns:

	28/10	4/11	11/11	18/11	25/11	2/12	9/12	16/12	23/12	30/12
Pneumonia:										
Cases ..	236	253	261	313	302	282	347	374	364	502
Deaths ..	141	198	177	188	207	198	236	289	351	383
Influenza:										
Deaths ..	17	19	20	16	24	28	34	33	54	102

Totals from the first week of the year in the Great Towns compared with the preceding two years are as follows:

	1948	1949	1950
Pneumonia:			
Cases	17,416	19,043	16,452
Deaths	10,337	11,786	10,461
Influenza:			
Deaths	608	3,090	1,859

The following Table shows notifications of pneumonia—primary and influenzal—in England and Wales:

	28/10	4/11	11/11	18/11	25/11	2/12	9/12	16/12	23/12	30/12
Pneumonia ..	435	433	465	558	490	497	573	684	647	962
Corresponding week, 1949	303	419	534	667	710	715	734	676	634	842

The following cities and large towns had the largest numbers of pneumonia notifications in the week ending December 30, 1950: London 64, Birmingham 38, Sunderland 32, Sheffield 24, Manchester 23, Liverpool 18, Gateshead 15, Salford 14, Newcastle 14.

The following weekly claims have been made on the Ministry of National Insurance: Week ending October 28, 1950, 127,245; November 4, 144,446; November 11, 148,497; November 18, 147,415; November 25, 145,783; December 2, 142,011; December 9, 141,403; December 16, 128,842; December 23, 87,277 (four days only).

Regional totals of influenza deaths in the Great Towns in the week ending December 30, 1950, were: Greater London (included in London and South-east) 22, Northern 34, East and West Ridings 4, North-western 30, North Midland 1, Midland 6, Eastern 1, London and South-east 24, Southern 2, South-western 3, Wales 1; total 106.

Infectious Diseases

A large fall occurred in the number of notifications of most infectious diseases in England and Wales during the week ending December 23, 1950. The falls included whooping-cough 768, scarlet fever 242, dysentery 190, and acute poliomyelitis 54. The only large exception to the general decline was a rise of 105 in the notifications of measles. The notifications of acute poliomyelitis were 7 fewer than in the corresponding week of 1947 and 18 fewer than in 1949. One-third of the cases of acute poliomyelitis were notified in London and the adjacent counties during the week.

During the 51st and 52nd weeks of 1950, 54 and 102 deaths were attributed to influenza in the Great Towns of England and Wales. These are about twice the numbers registered during the corresponding weeks of recent years. Notifications of influenza usually rise to a maximum towards the end of the first quarter, and it is possible that the present outbreak will be larger than those that have occurred in the last few years. The disease is now prevalent chiefly in the northern cities.

Dysentery

The notifications of dysentery were 190 fewer than in the preceding week, but with this exception they were the highest ever recorded. The chief features of the returns during the week were large falls in Leicestershire 138 and Lancashire 89 and rises in Staffordshire 49 and Middlesex 29.

The largest returns were Lancashire 173 (Southport C.B. 53, Oldham C.B. 25, Salford C.B. 20, Manchester C.B. 20); Leicestershire 101 (Leicester C.B. 89); Yorkshire West Riding 136 (Earby U.D. 58, Bradford C.B. 19, Sheffield C.B. 14); Staffordshire 74 (Stoke-on-Trent C.B. 44, Stafford M.B. 11, Cheadle R.D. 10); Middlesex 69 (Edmonton M.B. 54); London 60 (Poplar 13, Islington 12); Shropshire 48 (Shrewsbury M.B. 26, Whitchurch U.D. 12); Warwickshire 34 (Birmingham C.B. 30); Cheshire 29 (Wirral R.D. 8); Essex 26 (Romford M.B. 15, West Ham C.B. 9); Glamorganshire 23; Monmouthshire 23 (Newport C.B. 9); Surrey 22 (Esher U.D. 9); Yorkshire North Riding 17 (Middlesbrough C.B. 17); Northumberland 16 (Glendale R.D. 9); Devonshire 16 (Exeter C.B. 10); Durham 15; Norfolk 15 (Mitford and Launditch R.D. 15); Kent 13; Somersetshire 12; Sussex 11; Southampton 10; Northamptonshire 10 (Kettering M.B. 9); Gloucestershire 10.

Week Ending December 30

The notifications of infectious diseases in England and Wales during the week included: scarlet fever 1,026, whooping-cough 3,690, diphtheria 41, measles 17,468, acute pneumonia 962, acute poliomyelitis 53, dysentery 691, paratyphoid fever 6. Four cases of smallpox, including two imported cases, were notified. In the Great Towns the number of deaths from influenza was 102.

Quarterly Returns for Scotland

The birth rate during the third quarter of 1950 was 17.3 per 1,000, being 1.7 below the average of the five preceding September quarters. The infant mortality was 33 per 1,000 registered live births, and this rate was 2 below the rate for the third quarter of 1949 and 10 below the five-years average. The stillbirths were equivalent to a rate of 27 per 1,000 total births. The general death rate was 10.2 per 1,000 population and was 0.1 below the five-years average.

The death rate from all forms of tuberculosis was 43 per 100,000 and that from respiratory tuberculosis was 37. The former rate is 16, and the latter 14, below the rate for the corresponding quarter of 1949. Of the 36 deaths attributed to the principal epidemic diseases, 13 were due to influenza, 9 to measles, and 9 to whooping-cough; 38 deaths were registered from acute poliomyelitis.

Industrial Accidents and Diseases

According to the *Ministry of Labour Gazette* (December, 1950) the number of workpeople other than seamen in the United Kingdom who died from accidents in the course of their employment in November, 1950, was 151, compared with 112 (revised figure) in October, 1950, and 123 (revised figure) in November, 1949. There were 33 deaths underground in coal mines, and 18 in the railway service. The deaths of 11 seamen were reported.

The following cases of industrial diseases were reported in November, 1950: lead poisoning, 4; anthrax, 6 (5 of these were in the handling and sorting of hides and skins); epitheliomatous ulceration, 9 (pitch 2, tar 5, oil 2); and chrome ulceration, 10; total, 31.

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INFECTIOUS DISEASES AND VITAL STATISTICS

We print below a Summary of Infectious Diseases and Vital Statistics in the British Isles during the week ended December 23.

Figures of Principal Notifiable Diseases for the week and those for the corresponding week last year, for: (a) England and Wales (London included). (b) London (administrative county). (c) Scotland. (d) Eire. (e) Northern Ireland.

Figures of Births and Deaths, and of Deaths recorded under each infectious disease, are for: (a) The 126 great towns in England and Wales (including London). (b) London (administrative county). (c) The 16 principal towns in Scotland. (d) The 13 principal towns in Eire. (e) The 10 principal towns in Northern Ireland.

A dash — denotes no cases; a blank space denotes disease not notifiable or no return available

Disease	1950					1949 (Corresponding Week				
	(a)	(b)	(c)	(d)	(e)	(a)	(b)	(c)	(d)	(e)
Meningococcal infection	31	3	18	1	—	28	—	10	—	1
Deaths		1	1				—	1		
Diphtheria	46	2	13	5	1	81	15	25	1	3
Deaths	—	—	—	—	—	—	—	—	—	—
Dysentery	1,024	60	148		4	221	16	67	1	4
Deaths		—			—				—	—
Encephalitis, acute	6	1	—		—	1	—	—	—	—
Deaths		—		—			—			
Erysipelas			28	5	4			28	6	3
Deaths							—			
Infective enteritis or diarrhoea under 2 years				9	11				27	
Deaths	22		7	—	—	27	1	1	4	2
Measles*	14,328	1556	197	120	79	1,684	32	23	47	107
Deaths†		1	—	2	—			—	—	1
Ophthalmia neonatorum	37	1	9		1	35	2	7	—	—
Deaths										
Paratyphoid fever	3	—	—		1	5	1	2(B)	—	—
Deaths	—		—		—	—	—	—	—	—
Pneumonia, influenzal	647	42	12		2	634	29	7	4	7
Deaths (from influenza)‡	54	4	5	—	1	27	5	2	2	2
Pneumonia, primary	350	69	301	¶	12	296	44	296	22	9
Deaths				12					18	
Poliomyelitis, acute	53	6	4	3	—	81	8	2	2	—
Deaths§	3	1		—		5	—			
Puerperal fever			1				—	3		
Deaths										
Puerperal pyrexia‖	74	10	5		1	72	6	7	1	1
Deaths							—			
Relapsing fever	—	—			—	—	—			—
Deaths										
Scarlet fever	1,106	87	176	57	42	2,416	142	221	81	176
Deaths†		—	—	—	—			—	—	—
Smallpox	—	—	—		—	—	—	—	—	—
Deaths					—				—	—
Typhoid fever	5	1	—	2	—	3	—	—	1	1
Deaths	—	—	—		—	1	—	—	—	—
Typhus fever	—	—	—	—	—	—	—	—	—	—
Deaths					—				—	—
Whooping-cough	3,735	360	604	77	30	936	39	63	42	44
Deaths	13	2	—	—	—	3	—	—	1	—
Deaths (0–1 year)	299	39	42	7	10	337	43	42	48	12
Deaths (excluding stillbirths)	6,971	1085	791	188	154	5,712	862	701	255	149
Annual death rate (per 1,000 persons living)			15·9					14·1	15·8	
Live births	6,411	995	809	347	206	6,569	1015	877	326	204
Annual rate per 1,000 persons living			16·3					17·6	20·2	
Stillbirths	164	28	21			177	23	24		
Rate per 1,000 total births (including stillborn)			25					27		

* Measles is not notifiable in Scotland, and the returns are therefore an approximation only.

† Deaths from measles and scarlet fever for England and Wales, London (administrative county), will no longer be published.

‡ Includes primary form for England and Wales, London (administrative county), and Northern Ireland.

§ The number of deaths from poliomyelitis and polio-encephalitis for England and Wales, London (administrative county), are combined.

‖ Includes puerperal fever for England and Wales and Eire.

¶ Primary pneumonia no longer notifiable in Eire.

Medical News

New Year Honours

In the New Year Honours a Knighthood was conferred on Mr. Percy Barter, C.B., Chairman of the Board of Control, and on Mr. Thomas Dalling, M.R.C.V.S., Chief Veterinary Officer, Ministry of Agriculture and Fisheries. Mr. Barter has been Chairman of the Board of Control and Under Secretary, Ministry of Health, since 1945, having been previously secretary of the Board from 1930 to 1939 and Principal Assistant Secretary, Ministry of Health, from 1940 to 1945. Mr. Dalling has undertaken important work on canine distemper and on problems connected with immunization against other animal diseases, including tuberculosis.

Amended National Formulary

The second amendment list, dated 1950, has just been issued. The main change is the introduction of dexamphetamine (the official name for the proprietaries "dexedrine" and "dexamed") as Tabellae Dexamphetaminae Sulphatis, "Six tablets, each containing 5 mg., to be dispensed unless otherwise directed."

Profits from Streptomycin

Dr. Albert Schatz, of New York, who was a student of Dr. Selman Waksman when streptomycin was discovered, recently brought a successful court action against Dr. Waksman for recognition as co-discoverer and a share in the royalties. He will receive 3% of royalties accruing after October 1, 1950, and $150,000 dollars (about £43,500) for relinquishing foreign rights in the antibiotic. The net royalties up to this date are estimated at some $2,360,000 (about £843,000), but Dr. Waksman has received only about $350,000 (£125,000). The remainder went to the Rutgers Research and Endowment Foundation, to which Dr. Waksman made over part of the rights. It is not directly connected with Rutgers University, where the drug was discovered. Under the new settlement 7% of the net royalties will be divided among 13 other graduate students of Rutgers and cash bonuses will be paid to 12 assistants. The Foundation will still continue to receive 80% of the streptomycin royalties, and Dr. Waksman will receive 10%. Originally Dr. Waksman received 20%, but when the royalties began to mount up to big figures he asked the Foundation to cut his share by half.

New Journal of Therapeutics

The publication of a new monthly journal, *Antibiotic and Chemotherapy*, is announced by the Washington Institute of Medicine. Early contributions will include reviews of the present status of antibiotic therapy, A.C.T.H., cortisone, the chemotherapy of tuberculosis, and details of newer antibiotics. The journal is published from 1708, Massachusetts Avenue, Washington, at a subscription of $10 per annum.

In Brief

Dr. Edith Summerskill, Minister of National Insurance, is visiting Egypt, January 14–21, to study the development of social services there.

Wills

Dr. Herbert Morley Fletcher, formerly consulting physician, St. Bartholomew's Hospital, London, left £21,196, including £100 to the Royal Medical Benevolent Fund. Dr. Arthur Stanbury Cobbledick, formerly of Trevone, near Padstow, Cornwall, left £11,738, including £100 to the Royal Eye Hospital, St. George's Circus, London, S.E. Dr. Douglas Craig, of Corringham, Essex, left £27,000. Dr. Robert Percy McDonnell, late chief medical adviser, Department of Local Government and Public Health, Ireland, left £11,838. He left £50 for Masses and £25 each to the Dublin branches of the N.S.P.C.C. and S.P.C.A.

COMING EVENTS

Royal Institute of Public Health and Hygiene

Whole-time and part-time courses for the Certificate and Diploma in Public Health and for the Diploma in Industrial Health begin on various dates on and after January 26. Full details may be obtained from the secretary, 28, Portland Place, London, W.1.

Radiology Course

The Faculty of Radiologists will hold a postgraduate week-end course on February 17 and 18. Lecture demonstrations will be given on (1) diagnostic radiology and (2) radiotherapy. Full particulars may be obtained from the honorary secretary, Faculty of Radiologists, 45, Lincoln's Inn Fields, London, W.C.2.

Social and Preventive Paediatrics

A course of 10 lectures for postgraduates on social and preventive paediatrics began at the Hospital for Sick Children, Great Ormond Street, London, W.C., on January 8, when Sir James Spence spoke on "The Influence of Environment on Disease in Childhood." The remaining lectures will be delivered weekly on Mondays, at 5 p.m., until March 12, and details will be published in the "Societies and Lectures" column of the *Journal*. The fee for the course is £3 3s. (single lectures, 7s. 6d.), and applications for tickets of admission, accompanied by a remittance, should be sent to the secretary of the Institute of Child Health at the hospital.

Psychopathology of Cerebral Diseases

Professor Manfred Bleuler, of the University of Zurich, will deliver a lecture on "The Psychopathology of Cerebral Diseases" at the Institute of Neurology, National Hospital, Queen Square, London, W.C., on Tuesday, February 13, at 5 p.m.

Polish Doctors in Great Britain

An Assembly of Polish Doctors in Great Britain will take place at B.M.A. House, Tavistock Square, London, W.C., on Friday and Saturday, January 19 and 20. On January 19 the proceedings, which begin at 11 a.m., include an address by Sir Alexander Fleming, F.R.S., on "The Search for Antibiotics," and, in the afternoon, a paper by Dr. L. Surzynski on "Social and Professional Problems Facing Emigré Polish Doctors." Full particulars may be obtained from the chairman of the Polish Medical Association, 14, Collingham Gardens, London, S.W.5.

SOCIETIES AND LECTURES

Monday

DURHAM UNIVERSITY.—At Royal Victoria Infirmary, Newcastle-upon-Tyne, January 15, 5.15 p.m., "*Experiments in Learning*," Rutherford Morison Lecture by Professor J. Z. Young, F.R.S.

HUNTERIAN SOCIETY.—At the Mansion House, London, E.C., January 15, 8.30 p.m., "*The Arteriographic Approach to the Study of Arteritis*," by Professor F. Orban (Liége).

●INSTITUTE OF CHILD HEALTH, Hospital for Sick Children, Great Ormond Street, London, W.C.—January 15, 5 p.m., "*The Economics of Child Health*," by Dr. J. D. Kershaw.

INSTITUTE OF UROLOGY.—At St. Paul's Hospital, Endell Street, London, W.C., January 15, 10 a.m., "*Calculous Disease of Bladder and Urethra*," by Mr. F. J. F. Barrington; 11.30 a.m., "*Marion's Disease and Diverticulum of Bladder*," by Mr. H. K. Vernon; 2 p.m., "*Congenital Defects of Bladder and Urethra*," by Mr. J. E. Semple; 3.30 p.m., demonstration: *Endoscopic Instruments*, by Mr. A. R. C. Higham; 5 p.m., "*Administration of Sulphonamides and Antibiotics*," by Dr. A. H. Harkness.

ROYAL EYE HOSPITAL, St. George's Circus, Southwark, London, S.E.—January 15, 4 p.m., "*Optics (Revision)*," by Mr. H. N. Reed.

●ROYAL EYE HOSPITAL, St. George's Circus, Southwark, London, S.E.—January 15, 5.30 p.m., "*Anatomy of the Eye and Orbit*," by Professor T. Nicol.

SCIENTIFIC FILM ASSOCIATION: MEDICAL COMMITTEE.—At Royal Society of Medicine, 1, Wimpole Street, London, W., January 15, 5 p.m. Films on Child Health will be shown and discussed.

Tuesday

BRITISH POSTGRADUATE MEDICAL FEDERATION.—At London School of Hygiene and Tropical Medicine, Keppel Street, London, W.C., January 16, 5.30 p.m., "*Observations on the Circulation in the Human Limbs*," by Professor H Barcroft.

EUGENICS SOCIETY.—At Royal Society, Burlington House, Piccadilly, London, W., January 16, 5.30 p.m., "*Congenital Malformations*," by Dr. C. O. Carter.

●INSTITUTE OF DERMATOLOGY, Lisle Street, Leicester Square, London, W.C.—January 16, 5 p.m., "*Zoonoses (Parasitic Infections)*," by Dr. M. S. Thomson.

INSTITUTE OF UROLOGY.—(1) At St. Paul's Hospital, Endell Street, London, W.C., January 16, 10 a.m., "*Injuries, Benign Growths, and Foreign Bodies in the Bladder*," by Mr. Harland Rees; 11.30 a.m., "*New Growths of the Bladder (1)*," by Mr. J. G. Sandrey; 2 p.m., "*New Growths of the Bladder (2)*," by Mr. J. G. Sandrey. (2) At St. Peter's Hospital, Henrietta Street, London, W.C., 3.30 p.m., ward round by Mr. Harland Rees; 5 p.m., "*Histology of Bladder Growths*," by Dr. C. E. Dukes.

ROYAL EYE HOSPITAL, St. George's Circus, Southwark, London, S.E.—January 16, 5 p.m., "*Surgery of the Iris*," by Mr. T. M. Tyrrell.

SOCIETY FOR THE STUDY OF ADDICTION.—At Medical Society of London, 11, Chandos Street, Cavendish Square, London, W., January 16, 4 p.m., "*Clinical Experiences in the Treatment of Alcoholism*," by Dr. J. A. Hobson. A discussion will follow.

Wednesday

HARVEIAN SOCIETY OF LONDON, 11, Chandos Street, London, W.—January 17, 7.45 p.m., annual general meeting; 8.45 p.m., "*The Continuing Problem of Phthisis*," presidential address by Dr. R. R. Trail.

●INSTITUTE OF DERMATOLOGY, Lisle Street, Leicester Square, London, W.C.—January 17, 5 p.m., "*Medical Mycology*," lecture-demonstration by Dr. R. W. Riddell.

INSTITUTE OF UROLOGY.—At St. Paul's Hospital, Endell Street, London, W.C., January 17, 11.30 a.m., "*A Survey of the Methods of Treatment in Benign Prostatic Enlargement*," by Mr. A. C. Morson; 2 p.m., "*Hydronephrosis*," by Mr. H. P. Winsbury-White; 3.30 p.m., "*X-rays*," by Mr. H. K. Vernon; 5 p.m., *Museum Demonstration*, by Mr. D. I. Williams.

ROYAL EYE HOSPITAL, St. George's Circus, Southwark, London, S.E.—January 17, 5.30 p.m., "*Retinoblastoma*," by Mr. R. P. Crick.

ROYAL MICROSCOPICAL SOCIETY, Tavistock House South, Tavistock Square, London, W.C.—January 17, 5.30 p.m., annual general meeting; 6 p.m., "*The Virus and the Cell*," presidential address by Dr. G. M. Findlay.

Thursday

BRITISH INSTITUTE OF RADIOLOGY, 32, Welbeck Street, London, W.—January 18, 8.15 p.m., joint meeting with Radiological Section, Royal Society of Medicine, and Faculty of Radiologists. Symposium: "*The Reticuloses*," speakers, Drs. W. St. C. Symmers, R. Bodley Scott, and F. Campbell Golding.

BRITISH POSTGRADUATE MEDICAL FEDERATION.—At London School of Hygiene and Tropical Medicine, Keppel Street, London, W.C., January 18, 5.30 p.m., "*Cerebral Responses to Peripheral Stimuli in Man*," by Dr. G. D. Dawson.

EDINBURGH CLINICAL CLUB.—At B.M.A. Rooms, 7, Drumsheugh Gardens, Edinburgh, January 18, 8 p.m., "*Sinusitis and Nasal Catarrh*," by Dr. J. F. Birrell.

INSTITUTE OF UROLOGY.—At St. Paul's Hospital, Endell Street, London, W.C., January 18, 10 a.m., "*Retropubic Operations on the Prostate*," by Mr. A. W. Badenoch; 11.30 a.m., "*Endoscopic Operations on the Prostate*," by Mr. A. W. Badenoch; 2 p.m., "*Transvesical Operations on the Prostate*," by Mr. A. R. C. Higham; 3.30 p.m., ward round by Mr. D. I. Williams; 5 p.m., "*Radiotherapy and Radium*," by Dr. S. B. Osborn.

LIVERPOOL MEDICAL INSTITUTION, 114, Mount Pleasant, Liverpool.—January 18, 8 p.m., "*Inguinal Hernia*," by Mr. John A. Shepherd.

ROYAL EYE HOSPITAL, St. George's Circus, Southwark, London, S.E.—January 18, 5 p.m., "*Ocular Palsy*," by Dr. S. Nevin.

ROYAL SOCIETY OF TROPICAL MEDICINE AND HYGIENE.—At 26, Portland Place, London, W., January 18, 7.30 p.m., "*Foreami*" (*Fonds Reine Elisabeth pour l'Assistance Médicale aux Indigènes*) *in the Belgian Congo*, by Professor René Mouchet.

ST. GEORGE'S HOSPITAL MEDICAL SCHOOL, Hyde Park Corner, London, S.W.—January 18, 4.30 p.m., lecture-demonstration on neurology.

UNIVERSITY COLLEGE (PHYSIOLOGY THEATRE), Gower Street, London, W.C.—January 18, 5.15 p.m., "*Physico-chemical Properties and Pharmacological Activity of Drugs*," by Mr. F. Bergel, Ph.D., D.Sc.

Friday

BRITISH INSTITUTE OF RADIOLOGY.—January 19, joint meeting with Radiological Section, Royal Society of Medicine, and Faculty of Radiologists continued. (1) At Royal Society of Medicine, 1, Wimpole Street, London, W., 10.30 a.m., symposium: "*The Reticuloses*," speakers, Dr. Brian Russell, Professor R. McWhirter, Drs. D. G. Galton, J. D. N. Nabarro, and John Boland; (2) At Royal College of Surgeons of England, Lincoln's Inn Fields, London, W.C., 2.15 p.m., symposium: "*The Reticuloses*," speakers, Drs. W. M. Levitt, Gwen Hilton, and Geoffrey Boden; (3) At Dorchester Hotel, London, W., 7.30 p.m., dinner.

BRITISH INSTITUTE OF RADIOLOGY, 32, Welbeck Street, London, W.—January 19, 5 p.m., meeting of medical members.

INSTITUTE OF UROLOGY.—At St. Paul's Hospital, Endell Street, London, W.C., January 19, 10 a.m., "*Malignant Disease of the Prostate*," by Mr. J. D. Fergusson; 11.30 a.m., "*Stricture of the Urethra*," by Mr. J. E. Semple; 2 p.m., "*Infertility of the Male*," by Mr A. R. C. Higham; 3.30 p.m., *Museum Demonstration*, by Mr. J. E. Semple.

LONDON ASSOCIATION OF THE MEDICAL WOMEN'S FEDERATION.—At Royal Free Hospital School of Medicine, 8, Hunter Street, Brunswick Square, London, W.C., January 19, 8.30 p.m., "*Recent Developments in Cardiac Surgery at Guy's Hospital*," by Mr. Lawrence Whytehead.

●ROYAL EYE HOSPITAL, St. George's Circus, Southwark, London, S.E.—January 19, 5.30 p.m., "*Physiology of the Eye*."

ROYAL MEDICAL SOCIETY, 7, Melbourne Place, Edinburgh.—January 19, 8 p.m., "*The Curious Effects of Carbon Monoxide Poisoning*," address by Dr. J. K. Slater.

Saturday

NUTRITION SOCIETY.—At London School of Hygiene and Tropical Medicine, Keppel Street, Gower Street, London, W.C., January 20, 10.30 a.m., "*The Comparative Merits of Animal and Vegetable Foods in Nutrition*." Discussion, speakers, Dr. Tristram (St. Andrews), Dr. Carpenter (Rowett Institute), Dr. Blaxter (Hannah Dairy Research Institute), Dr. Yates (Rothamsted Institute), Dr. Lucy Wills (Royal Free Hospital), Dr. Harriette Chick (Cambridge), and Dr. Deane (Cambridge).

SOUTH-EAST METROPOLITAN REGIONAL TUBERCULOSIS SOCIETY.—At Chest Clinic, Health Department, Town Hall, Walworth Road, London, S.E., January 20, 10.30 for 11 a.m., general meeting. Clinical programme as follows: "*Tuberculous Conditions of the Skin*," by Dr. W. G. Tillman; "*Oral Conditions in Pulmonary Tuberculosis*," by Dr. G. E. King-Turner; discussion: "*Domiciliary Treatment of Pulmonary Tuberculosis*," by Dr. E. K. Pritchard. At Brunswick Tavern, Old Kent Road, London, S.E., 1.30 p.m., luncheon.

BIRTHS, MARRIAGES, AND DEATHS

BIRTHS

Bulstrode.—On January 5, 1951, at Hartwell House, Hartfield, Sussex, to Jacqueline (formerly Kent), wife of Dr. J. C. Bulstrode, a son.

Habibis.—On December 23, 1950, to Stella Murray, M.B., B.S., D.C.H., wife of Dr H. S. Habibis, a son.

Hughes.—On January 1, 1951, at Torbay Hospital, Torquay, to Joy (formerly Laxton), wife of Dr. D. R. Hughes, a second son—Richard John.

Murphy.—On December 30, 1950, to Inez, wife of Dr. E. F. Murphy, Northowram Hall, Halifax, a daughter.

Sears.—On December 31, 1950, at Hammersmith Hospital, to Dr. Janet Sears (formerly Conn), wife of Dr. H. T. N. Sears, a daughter.

Thurston.—On January 3, 1951, in Trinidad, to Gay, wife of Dr. John V. Thurston, a sister for Graham—Sheila.

Wells.—On January 1, 1951, at Cardiff, to Barbara (formerly Sharratt), wife of Albert L. Wells, M.B., D.C.P., Llandough House, Penarth, Glam, a son.

MARRIAGES

Goodson—Taylor.—On December 26, 1950, at the Parish Church, Tacoma, Washington, U.S.A., Michael Piers Goodson, M.A., M.B., B.Chir., to Leonora Taylor, S.R.N., both of London.

DEATHS

Bertram.—On December 16, 1950, at his home, "Masons," Ilton, Ilminster, Somerset, Drummond Rewcastle Forbes Bertram, O.B.E., M.B., B.S., D.M.R., Surgeon Commander, R.N., retired, aged 47.

Cathcart.—On January 4, 1951, at South Court Nursing Home, Broadstairs, George Clark Cathcart, M.D., of 11, Upper Wimpole Street, London, W., aged 90.

Cooper.—On December 21, 1950, William Francis Cooper, M.B., B.Ch., late of Hertcombe, Coombe Warren, Kingston, Surrey.

Coutts.—On December 19, 1950, at 47, Rouge Bouillon, St. Helier, Jersey, C.I., Duncan Coutts, M.D., F.R.C.O.G., Lieutenant-Colonel, I.M.S., retired, aged 68.

Fraser.—On December 22, 1950, at Pitlochry, Donald Alexander Fraser, M.B., Ch.B., D.P.H.

Fraser.—On January 2, 1951, at 16, Albyn Place, Aberdeen, Thomas Fraser, C.B.E., D.S.O., T.D., LL.D., M.B., Ch.B., D.P.H., aged 78.

French.—On January 1, 1951, at Guy's Hospital, London, S.E., Herbert Stanley French, C.V.O., C.B.E., M.D., F.R.C.P., of Cudworth Manor, Newdigate, Surrey, aged 76.

Girdlestone.—On December 30, 1950, Gathorne Robert Girdlestone, D.M., F.R.C.S., of Frilford Heath, Berks, aged 69.

Gully.—On December 28, 1950, at Du Cane Court, London, S.W., Robert Cullum Gully, M.D., formerly of St. George's Road, Southwark, London, S.E.

Harris.—On December 26, 1950, at 105, Whipps Cross Road, Leytonstone, London, E., Sampson George Victor Harris, M.D., D.P.H., aged 85.

Heath.—On December 23, 1950, at Barmouth, Merioneth, John Rippiner Heath, M.R.C.S., L.R.C.P., aged 63.

Linton.—On December 22, 1950, at a nursing-home, Glasgow, Robert Fenwick Linton, M.B., Ch.B., Lunan Lodge, Renfrew.

Macmillan.—On December 24, 1950, Hugh Agnew Macmillan, M.C., M.D., of The Cairnons, Methil, Fife.

Martin.—On December 27, 1950, at Elford, Douglas Avenue, Exmouth, Claude Buist Martin, C.M.G., M.B., C.M., Colonel, A.M.S., retired, aged 81.

Any Questions?

Correspondents should give their names and addresses (not for publication) and include all relevant details in their questions, which should be typed. We publish here a selection of those questions and answers which seem to be of general interest.

Treatment of Psychoses with A.C.T.H. or Cortisone

Q.—*Is treatment with adrenocorticotropic hormone (A.C.T.H.) or cortisone likely to help a patient suffering from a manic-depressive psychosis?*

A.—Treatment of manic-depressive psychosis by A.C.T.H. or cortisone is still in the experimental stage. It has been observed that in mentally healthy people injection of large amounts of these substances produces undesirable mental changes. It would therefore be risky to treat those already mentally ill by this means unless full precautions were taken, and it would be unjustifiable except as part of a controlled experiment. A number of psychotic patients have, however, with due regard for these points, been treated in the United States by investigators primarily concerned with the response of the adrenal cortex to stress in certain mental disorders. M. D. Altschule[1] and his colleagues in Boston have found that patients with schizophrenia and severe depression show greater than normal changes in carbohydrate metabolism after the injection of A.C.T.H., and their eosinophil count, uric acid, and 17-ketosteroid excretion are not diminished as they would be in non-psychotic persons given A.C.T.H. These investigators record unsatisfactory results from the clinical standpoint: in a depressed elderly woman the A.C.T.H. caused hallucinations to develop, and she was still extremely agitated and depressed after the administration of 570 mg. of A.C.T.H. spread over 17 days; a younger woman, also depressed, became rather worse after 900 mg. of A.C.T.H. spread over 10 days; others became more tense and restless. In Montreal another group of workers,[2] headed by R. A. Cleghorn, has administered A.C.T.H. to manic as well as to depressed patients, chiefly to study abnormalities of adrenal cortical function in manic-depressive disorders. In the depressed patients a slight initial improvement in their mental state was followed by some increased tension: any improvement disappeared quickly when the injection of A.C.T.H. was stopped. A manic patient treated by Cleghorn with cortisone for two weeks improved, so long as the cortisone was being given, but it could not safely be concluded that the cortisone was directly responsible for this. In an investigation in New York City, J. J. Smith[3] likewise found that A.C.T.H. in large quantities had not effected any clinical improvement in a few patients with agitated depression and mania whom he treated.

REFERENCES

1 *Arch. Neurol. Psychiat., Chicago*, 1950, **64**, 641.
2 *Canad. med. Ass. J.*, 1950, **63**, 325 and 329.
3 *Quart. J. Stud. Alcohol*, 1950, **11**, 190.

Persistent Urethral Discharge

Q.—*A patient of mine has had a sero-purulent urethral discharge for the last five months. Repeated bacteriological examination confirms the absence of gonococci, but there is a mixed flora of non-haemolytic streptococci, diphtheroids, and small Gram-negative bacilli. Penicillin and the sulphonamides have failed to clear up the discharge. What further investigations and treatment would you suggest, and is there any likelihood of a subsequent urethral stricture developing?*

A.—Before answering the query it should be emphasized that thorough cleansing of the meatus with spirit should precede the taking of specimens for microscopical examination. Most non-gonococcal urethral discharges are abacterial, but inadequate cleansing often leads to the mistaken diagnosis that one or more of the normal saprophytic flora is responsible for the infection. A bacterial (non-gonococcal) urethritis is extremely rare, such cases as there are being either due to mechanical causes (usually the insertion of foreign bodies) or secondary to infections of the upper urinary tract.

The patient in question is in all probability suffering from an abacterial urethritis. In these cases there is often a history of intercourse during or near a period. A possible explanation of this is that at such times the cervical canal opens up and becomes more vascular, and thus allows much more epithelium (containing the infective agent) to be desquamated into the vagina. Such infections are always resistant to penicillin and the sulphonamides, but "aureomycin" 0.5 g. eight-hourly by mouth for three days effects a cure in at least 80% of cases. Urethral stricture is a common sequel when the disease is of long standing. Stricture, which is never serious, can be diagnosed only by urethroscopy (it is called "urethroscopic stricture"), as there is no diminution in the calibre of the urethra. On the other hand, if the infection is bacterial, hard strictures, as in gonorrhoea, may occur. If further laboratory investigations show that the condition is bacterial in origin a thorough investigation of the upper urinary tract should be carried out by a urologist.

Haemolytic Disease of the Newborn

Q.—*I would be glad of your advice on the following case: the mother is Group O, Rh negative; the father is Group O, homozygous, Rh positive (type Rh_1Rh_2—CDe/cDE). The only child is Group O, Rh positive (type Rh_2rh—cDE/cde). The second pregnancy proceeded normally, antibodies developing in low titre at the 26th week. The titre gradually rose until the 35th week, when they were 1/512 in serum-albumin. No blocking antibodies ever developed. At the 36th week the titre doubled, but induction of labour was not carried out. A few days later the child died in utero. The patient has been told that in all future pregnancies the child will be affected and may die in utero, or if born alive will require exsanguination transfusion. If this is correct, can anything be done to prevent an antibody response? Is hapten of any use? If treatment will not prevent foetal damage, should the patient be allowed to become pregnant again?*

A.—The question does not give as full details as would be desirable. If the parents concerned have the commonest genotypes of the phenotypes stated—i.e., that the mother is rhrh (cde/cde) and the father Rh_1Rh_1 CDe/cDE) and that the antibody is anti-D—then any future pregnancy must be expected to yield a child suffering from haemolytic disease.

At the present time no method is known by which the maternal antibody response can be prevented or the foetus protected from the antibodies. The extracts which it was thought might have contained Rh hapten have not given satisfactory results in clinical trials, and their action *in vitro* is thought to be non-specific. Such other substances as have been tried have been ineffective, with the possible exception of methionine, which was thought to be of value by N. W. Philpott, M. Hendelman, and T. Primrose (*Amer. J. Obstet. Gynec.*, 1949, **57**, 125). Their results have not as yet been confirmed by other workers.

The procedures which should be considered in a future pregnancy are induction of labour and replacement transfusion. The table below shows the results of such treatment in 46 Rh-positive pregnancies in which the mothers had borne at least one previous child affected with haemolytic disease. The plan of treatment was to induce labour surgically at 36 weeks or as soon afterwards as the foetus was considered large enough, a minimum weight of 5 to 5½ pounds (2.26 to 2.48 kg.) as estimated by the obstetrician being required before induction was allowed. All except

the mildest cases of haemolytic disease received a replacement transfusion of one pint of compatible blood within a few hours of birth. The indications for replacement transfusion cannot as yet be laid down clearly, but as a general rule any child with a positive direct Coombs test and a cord haemoglobin level of under 15½ g. per 100 ml. should receive one. Although the series quoted is small, it shows that the prognosis for such a pregnancy is not hopeless.

	Stillborn	Neonatal Death	Survived
Results of latest pregnancies of 19 mothers whose previous affected infant was stillborn	5	5	9*
Results of latest pregnancies of 27 mothers whose previous affected infant was born alive	5	5	17

* Includes one case of kernicterus

The problem of allowing further pregnancies is one for the parents to consider after they have been informed of the probable risk. If, as is generally the case, they decide that a further pregnancy is undesirable, then advice on contraception should be given. In the uncomplicated case there are no grounds for advising either sterilization or termination of pregnancy.

Dupuytren's Contracture

Q.—*How should Dupuytren's contracture be treated? Is vitamin E of value in this condition?*

A.—The treatment of Dupuytren's contracture is still entirely an operative one. The classical methods of subcutaneous fasciotomy and excision of the affected fascia are still the two basic procedures. In the past it was customary to use fasciotomy for the mild cases and excision of the fascia for the severe cases. The present attitude is the reverse. The mild or moderate cases are those best suited to excision of the fascia, and in these an excellent result can be expected. In the severely contracted cases fasciotomy is performed to explore the possibility of treatment by excision. In most of them excision is of no value, as the joints of the finger are commonly stiff or subluxated. If a tentative fasciotomy shows this to be the case no operative treatment other than amputation is to be advised. In the operative excision of the fascia it is imperative to remove all skin which is involved in the Dupuytren process. Closure of the skin by means of a Z-plasty is a very valuable adjunct to the operation.

It is almost certain that vitamin E is valueless in this condition. Various writers have reported success with heavy doses of this vitamin (see *British Medical Journal*, 1949, **2**, 1399), while others (for example, A. R. King, *J. Bone Jt Surg.*, 1949, **31** B, 443) have reported equal failure. Many of the improvements were of subjective type, the patients saying that the hands felt softer or the fingers felt straighter, and that the aching pain had gone. Some observers have reported objective improvement in the extension of the fingers, others have reported failure of improvement in objective signs. One writer comments that in 12 cases out of 13 no value was seen with treatment at full dosage for six to eight weeks at a cost of approximately £5 per week.

Adiposis Dolorosa

Q.—*Is anything known of the aetiology and treatment of adiposis dolorosa?*

A.—There is no pathological basis for the differentiation of adiposis dolorosa from other forms of adiposity, although clinically the name is used if the adipose deposits tend to be painful and the patient depressed or melancholic.

The question therefore involves discussion of the whole complex problem of adiposity. This also applies to treatment, which has been dealt with many times in these columns, and broadly speaking involves three lines of approach: dieting, and the use of appetite depressors and diuretics. Thyroid is of limited value, and there is no hormone known which specifically catabolizes fat.

Urticaria in 8-week-old Twin

Q.—*I have under my care a baby of 8 weeks, one of identical twins. He was quite well until two weeks ago, when an urticarial rash developed and he began vomiting; he was also constipated. Examination showed that the colon was loaded with hard faeces, and treatment of the constipation cured the vomiting. However, the urticaria has continued, the hands have been grossly swollen, and huge weals have appeared on the face and trunk. On the assumption that the urticaria might be due to woollen clothes or to the detergent these were washed in, the child was given cotton underclothes washed in soapflakes only. In spite of this the condition has persisted, and now one leg is grossly swollen. The other twin, although having the same food (national dried milk) and living in the same environment, is quite well and is now two pounds heavier than his brother. Can you tell me: (1) What is the youngest age at which urticaria occurs? (2) Does the fact that one twin has reacted differently to the same condition prove that the twins are not identical? (3) Is it safe to give antihistamine drugs to children of 8 weeks, and, if so, what is the dosage?*

A.—The answers to the three questions are: (1) Urticaria may appear at any age, and has been reported in the newborn. (2) A different reaction to the same condition in twins does not prove that they are not identical, because other factors may not be the same, but in general one would expect identical twins to react in the same way. (3) The antihistamine drugs at present on the market can safely be given to infants, who seem to tolerate them better than most adults. The dose varies according to the drug used, but those for which a single adult dose is 25 mg. can be given in 10-mg. doses up to a total daily maximum equal to about 3 mg. per pound (0.45 kg.) of body weight.

It is rather unusual for an allergic reaction to cause such persistent swelling of the limbs. Is it possible that some other condition is present as well as an allergic reaction with urticaria? Vomiting and urticaria in a bottle-fed baby suggest a milk allergy, although colic and loose, fatty, or undigested stools would be expected rather than constipation. It is important to find the allergen before exhibiting antihistamine drugs, and it would be best to place the child on a boiled water, or glucose-water, diet for a couple of days to see if the symptoms disappear. If they do, then it would be wise to feed the child upon a soya-bean preparation before attempting to desensitize him to cow's milk. The delay in the appearance of symptoms does not rule out the possibility of sensitivity to cow's milk.

NOTES AND COMMENTS

Correction.—In the obituary notice of Sir Leonard Parsons, which was published in the *Journal* of December 30, 1950, it was wrongly stated that Sir Gilbert Barling was co-editor with Sir Leonard Parsons of *Disease in Infancy and Childhood*. It was Mr. Seymour Barling who was co-editor of this textbook.

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